Escherichia coli O157:H7

Issues and Ramifications

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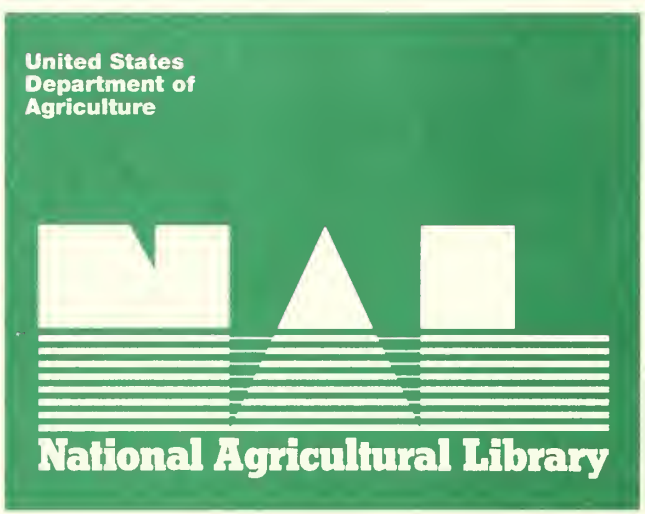


Contents

Executive Summary

The primary purpose of this report is to help define the role of cattle as a source of *E. coli* O157:H7 in food products. Although different modes of transmission from cattle to humans are discussed, the report concentrates on the vehicle most frequently implicated in human disease outbreaks, ground beef. The summary is divided into four sections: (1) Why the interest in *E. coli* O157:H7?, (2) What is known about *E. coli* O157:H7 in cattle?, (3) Do production and consumption patterns for ground beef offer any additional insight into *E. coli* O157:H7?, and (4) Future directions.

Why the interest in *E. coli* O157:H7?

Escherichia coli O157:H7 (O157) was first identified as a human pathogen capable of causing foodborne illness in 1982. However, the public was generally unaware of the existence of O157 until a decade later. In late 1992, an outbreak associated with the consumption of undercooked hamburgers began in Washington state. The more than 600 illnesses and the subsequent deaths of 4 children were publicized throughout the country. In addition, evidence suggesting that the frequency of O157 illness in humans is increasing has heightened concern. Of the 32 outbreaks reported in the U.S. from 1982 through 1993, 13 occurred in 1993.

Human illness associated with O157 is infrequent in comparison to illness associated with some other foodborne pathogens such as *Salmonella*. However, the range in severity of clinical illness and the potential for debilitating complications and death makes O157 a noteworthy food safety issue. The abdominal cramping and bloody diarrhea typical of O157-associated disease result from toxin production and subsequent destruction of the mucosal lining of the colon. In most patients, the disease is self-limiting. However, a small percentage of O157 cases progress to hemolytic uremic syndrome (HUS) and/or thrombotic thrombocytopenic purpura (TTP). The elderly and children less than 5 years old are at highest risk of developing these complications. Such cases may result in kidney failure or death.

Although not definitively established, it is believed that O157 inhabits the lower intestine of cattle and is shed in the feces. Human infection with O157 occurs primarily through ingestion of food contaminated with fecal material. Another recognized source of infection is O157-contaminated water. Human-to-human and calf-to-human transmission have also been documented.

Although O157 is one of many serotypes of a common and ubiquitous bacteria, a unique characteristic of O157 is the organism's hardiness. It can survive for extended periods in water, meat stored at subfreezing temperatures, acidic environments, and soil. The organism is, however, destroyed by thorough cooking or pasteurization.

A variety of foods have been implicated in O157-associated illnesses. Of the 24 outbreaks associated with foods, 17, or 71 percent, have been linked to bovine products. Contaminated ground beef was associated with 12 of the outbreaks, raw milk and roast beef with 2 each, and 1 with hot dogs containing beef. Cross-contamination of other foods, including apple cider, vegetables, and mayonnaise, by manure or meat products has been confirmed or is suspected in the seven other foodborne outbreaks.

Although not directly linked to human illness, several other meat and poultry products have been sampled for O157. In addition to beef, the organism has been isolated from veal kidneys, poultry, pork, and lamb. However, cross-contamination of these meat products is considered likely. To

date, farm-level testing in the U.S. has concentrated on cattle. As a result, the status of O157 in other food animal species is not known.

There is no definitive evidence of a geographic pattern of human O157 cases. However, a 2-year study concluded that a significantly higher percentage of stool samples were O157-positive from hospitals in the northern and western U.S. than in the southern part of the country.

Both O157 sporadic cases and outbreaks have a definite seasonal pattern. The four largest studies in the U.S. have revealed that at least 67 percent of sporadic cases occurred between May and September, with a peak in July and August. Of all U.S. outbreaks associated with O157, 88 percent have occurred from May through November.

At least 16 countries on 6 continents have documented human cases or bovine isolates of O157, indicating the widespread presence of the organism. Outside of the U.S., most occurrences of O157 illness have involved sporadic cases; only Canada and the United Kingdom have reported outbreaks. As in the U.S., cases have generally peaked in the summer and fall months.

What is known about *E. coli* O157:H7 in cattle?

The epidemiologic link between human O157-associated illness and products of bovine origin has raised many questions concerning the occurrence of the organism in the cattle population. Beyond the observation that O157 is not known to cause clinical disease in cattle under natural conditions, little is known about the on-farm ecology of the organism. Analysis of O157 on-farm studies indicates that virtually all types and breeds of cattle should be viewed as potential sources of O157 contamination. Changes in various management practices which may have allowed or enhanced the ability of the O157 organism to inhabit the gastrointestinal tract of cattle are under investigation. At present, no definitive cause and effect relationships have been established.

The only nationwide on-farm study completed to date focused solely on preweaned dairy heifers (National Dairy Heifer Evaluation Project, NDHEP). Other studies, primarily in Washington state, have looked at adult dairy and beef cattle, as well as dairy calves. All studies found relatively low percentages of cattle shedding O157 (animal prevalence), generally less than 1.0 percent. In the one study which has looked at beef premises, the prevalence of shedding among adult beef cattle was slightly higher than has been found among adult dairy cattle. In all studies, dairy heifers and calves generally had a higher prevalence of O157 shedding than did adult dairy cattle.

The prevalence of herds with O157 (herd prevalence) has generally been higher than the overall animal prevalence of O157. To date, in studies of premises not associated with O157 tracebacks, 27 (2.4%) of 1,139 dairies and 4 (16.0%) of 25 beef premises have been culture-positive for O157. However, research suggests that the greater the number of animals sampled on a premises, the greater the likelihood of finding that premises positive for O157. Because there have not yet been many studies that sampled more than a few animals per premises, it is probable that true herd prevalence in the U.S. is much higher than has been found to date. In addition, the NDHEP found no geographic patterns or regional differences in herd prevalence or overall animal prevalence.

Most of the initial prevalence studies have been based on one-time fecal sampling. Consequently, little is known concerning the carrier status of individual animals. Preliminary evidence does, however, suggest that cattle transiently or sporadically shed O157 in their feces and that the excretion period ranges from hours to weeks. These observations are important in that on-farm sampling of individual animals may not be an accurate reflection of the shedding status of animals entering the slaughter facility.

Evaluation of seasonal patterns in the detection of O157-positive animals is inconclusive. One Washington state study found the isolation rate of O157 to be highest during the summer months, reaching a peak in September with 13 positives per 1,000 samples. This seasonal pattern was observed in both years of the study. These results are of interest in light of the seasonal pattern evident in human O157-associated illness. In contrast, no seasonal pattern could be established from the NDHEP, which had a much larger sample size and in which roughly equal numbers of dairy calves were sampled during each calendar month. The conflicting results may be attributable to differences in age, since the Washington study included adult cattle whereas the NDHEP did not.

No evidence was found of significant O157 transmission between dairy cattle in the NDHEP. Positive and negative herds were compared with respect to calf contact with older cattle and time spent by calves in maternity pens. Prevalence of O157 among preweaned dairy calves having contact with older heifers was similar to that of calves having no contact. No significant difference in herd prevalence was identified between herds that did and those that did not permit contact among calves and older animals. The length of time calves remained in the maternity area was likewise not shown to affect the prevalence of O157.

Various management and feeding practices are being examined for possible links to the presence of O157. Several practices have been found to have either a positive or negative association with the presence of O157 (Table 1). Whether or not these associations are relevant to the colonization of cattle with O157, or if cattle are even truly colonized by O157, is not yet known.

Feeding subtherapeutic levels of antibiotics to cattle to improve feed conversion and rates of weight gain is a management practice that has raised concerns. No evidence exists to suggest that O157 has acquired resistance to antibiotics. In fact, the opposite is true; most O157 organisms are susceptible to a variety of antibiotics. In addition, the use of antibiotics in cattle feed has been reduced since 1985. Current estimates indicate that only about 10 percent of all feed produced for beef cattle in the U.S. is formulated to contain antibiotics.

There is speculation that the use of ionophores, a class of antibiotics which is currently fed to certain types of cattle, may have allowed or enhanced the ability of O157 to become established as part of the intestinal microflora of cattle. The approval and subsequent adoption of ionophores for feedlot diets of cattle in the mid- to late-1970's roughly coincides with the identification of O157 as a foodborne human pathogen. Ionophore products are currently reported to be used in the diets of more than 90 percent of feedlot and farm-fed cattle and in less than 50 percent of replacement heifers and beef and dairy calves. Ionophores have been shown to inhibit gram-positive organisms in the rumen and, therefore, may allow the increased proliferation of gram-negative organisms such as *E. coli*. One study has reported that dairy farms feeding ionophores in grain had a higher O157 prevalence in calves than did farms not feeding ionophores. However, a follow-up study found no such association.

Other management practices can result in increased levels of stress in cattle. Weaning, abrupt changes in dietary composition, fasting, shipping, disease, or changes in immunologic status can predispose animals to shifts in the normal microflora of the gastrointestinal tract. It has been suggested that these shifts may result in increased numbers and/or increased shedding of O157 in cattle.

Dietary stress may be an especially important factor. The first notable dietary stress in an animal's life is weaning. One study of dairy calves revealed that the prevalence of O157 in postweaned calves was three times higher than among preweaned calves. Studies in nonbovine species have shown increased numbers of *E. coli* organisms in the intestinal tract post-weaning. *E. coli* numbers

have also been shown to increase in the gastrointestinal tracts of adult animals and birds following starvation or abrupt dietary changes. Cattle are usually held off feed in the hours prior to slaughter.

Transportation provides another source of stress for livestock and may be a critical factor prior to slaughter. There is some indication, based on a recent survey of packers, that transport distances to slaughter are greater for cows and bulls than they are for fed steers and heifers and have increased over the past 10 years. Whether greater transport distance leads to increased stress is not known, but greater time in transport and holding has been shown to increase rates of infection of cattle with organisms such as *Salmonella*.

Although there has been speculation that mastitic cows may be a primary source of O157 contamination, no evidence exists to single out this particular subgroup of the cattle population. No O157 was identified in more than 500 cases of coliform mastitis in 2 separate 1993 studies conducted in California and Pennsylvania. In addition, patterns in the recorded cases of clinical mastitis identified at slaughter do not correspond to trends in outbreaks and sporadic cases of human O157-associated illness. Neither the number nor the rate of mastitic cows at slaughter increased between 1983 and 1992.

Similarly, no evidence has been presented which argues for focusing on nonambulatory cows (downer cows) as a major source of O157. The hypothesis that possible increased antibiotic usage in nonambulatory cattle could help to select for O157 or allow O157 to more readily colonize such animals does not seem highly plausible since O157 is itself susceptible to most antibiotics. However, increased stress as a result of the downer condition may increase the likelihood of shedding O157 if it were present. A current Food Safety and Inspection Service study of nonambulatory cattle should help define any relationship between O157 and such animals.

Do production and consumption patterns for ground beef offer any additional insight into *E. coli* O157:H7?

Contaminated ground beef has been the most frequently identified vehicle for O157 in human disease outbreaks. The introduction of O157 may occur at any point along the entire production to consumption continuum. Therefore, changes in the continuum over the past decade need to be identified and examined for potential impacts on ground beef contamination or increased human exposure to O157-contaminated ground beef.

Relative proportions of different types of cattle slaughtered in the U.S. have changed little since 1980. Steers and heifers accounted for approximately 80 percent of cattle slaughtered, cows 18 percent, and bulls 2 percent. Calf slaughter was minimal when compared with cattle slaughter and meat from calves generally does not go into ground beef. Production for all types of cattle continued to concentrate geographically into fewer and larger herds, particularly in dairy and cattle feeding operations.

Marketing of all types of cattle for slaughter has changed somewhat over the same time period. Currently on a national basis, greater percentages of cattle are being sold directly to packing establishments rather than being marketed indirectly through public markets. In 1980, 88 percent of steers and heifers and 35 percent of cows were sold directly, but by 1990 those figures were 94 and 40 percent, respectively.

Slaughter facilities have become larger and more concentrated geographically, particularly in the Great Plains region. In 1992, 90 percent of all fed steers and heifers were slaughtered in only 33

plants, as compared to 90 plants in 1983. In 1992, 90 percent of cows were slaughtered in 68 plants, down from 152 plants in 1983.

Once cattle have been slaughtered, ground beef production flows through a variety of processing and distribution channels (Figure 1). Ground beef is produced directly in some slaughter plants from varying combinations of cuts and trimmings produced in-house, purchased trimmings, and domestic and imported boneless manufacturing-grade beef (BMB). Ground beef is also produced by grinders and retailers who purchase carcasses, boxed beef, bulk trimmings, and/or coarse ground trimmings from slaughter plants, other grinders, and/or distributors. There are currently 2,965 grinders in the U.S., of which less than 900 slaughter cattle. In 1992, there were 30,700 supermarkets with in-house meat departments.

The sale of fed beef by packers in the form of boxed beef rather than carcasses has steadily increased over the past 20 years and has had an impact on the production and distribution of ground beef. Boxed beef is sold as vacuum-packaged primal and subprimal cuts from which much of the bone and excess fat has been removed. This has meant that more trimmings from fed cattle are produced centrally at the slaughter plant rather than locally at the grinder or retail level.

The percentages of ground beef derived from individual types of cattle can be estimated as a national average for a given time period. In 1980, steers and heifers accounted for 56 percent of domestic raw product going into ground beef, cows for 36 percent, and bulls for 8 percent. By 1992, these percentages had changed only slightly to 58 percent steers and heifers, 34 percent cows, and 8 percent bulls. Boneless manufacturing beef imports also remained stable over the last decade, comprising approximately 15 percent of the total U.S. ground beef supply.

Although the proportion of cattle types slaughtered varies regionally, ground beef formulation does not. The formulation of ground beef is based largely on fat content. Lean meat from cows and bulls and lean and fat trimmings from fed steers and heifers can be shipped to various locations and then mixed to produce the final ground beef product.

The composition of ground beef in terms of the sources of raw product (lean and fat) appears to be independent of the production and distribution channel through which it passes. Any given pound or patty of ground beef can contain any combination of domestic cow meat, domestic fed beef, and/or imported BMB, regardless of the channel through which it was produced.

Per capita ground beef consumption (net disappearance) has increased since 1980 but is still below mid-1970 levels. Both the proportion of people that consumed ground beef in the form of hamburgers and the amount consumed increased in most age groups, including those at highest risk for O157-related illness, young children and the elderly. There was a corresponding increase in food expenditures outside of the home during the same time period. In 1992, fast food hamburgers accounted for about 47 percent of fast food sales, or 15 percent of all hotel, restaurant, and institution (HRI) sales.

Ground beef consumed in HRI settings, especially fast food establishments, is purchased primarily from grinders in the form of patties. Retail sources of ground beef are more evenly distributed among cow packers, fed beef packers, grinders, and trimmings produced in-house. This information along with the apparent increased consumption of hamburgers in HRI settings appears to indicate that a greater proportion of ground beef is now flowing through the channel from grinders to HRI's than during the early 1980's.

Future directions

- Would a geographic pattern in the number of O157 cases in humans tell something about O157 prevalence in cattle?

It is unlikely that any geographic pattern of human disease would reflect a geographic variation in the source of the O157-contaminated ground beef. In many cases the location of consumption of ground beef is not related to the original location of the sources of that ground beef nor to the potential sources of O157 contamination. Cattle that go into ground beef production may be moved great distances in the hours prior to slaughter, lean and fat trimmings may be shipped some distance prior to final grinding and mixing, and the final product may in turn be widely distributed.

- How can we explain the seasonality of human cases and outbreaks associated with ground beef?

The seasonality of cases and outbreaks associated with ground beef might be a reflection of any one or a combination of factors. First, there may be greater shedding of O157 by cattle during warmer months of the year, which may lead to increased contamination of ground beef during these months. Second, consumption of ground beef is higher during warmer months (summer barbecues, picnics, etc.). Third, there may be a greater likelihood of temperature abuse and/or less thorough cooking of ground beef during these months.

- Is there a particular channel in the ground beef production continuum that is associated with an increased risk of O157 contamination?

Ground beef intended for both retail and HRI can pass through various channels which may include a number of different steps. Although additional handling creates more opportunities for cross-contamination, no one channel can be singled out at this time as posing a greater risk.

- Should the goal be to eradicate O157 on the farm?

It does not currently appear feasible to target on-farm eradication of O157 for the following reasons: the lack of knowledge about the ecology of O157, the widespread geographic distribution of the organism, the fact that O157 has been found in both beef and dairy cattle, and the difficulty of identifying infected animals because of the likelihood of sporadic shedding and the absence of clinical disease. Since the risk of O157 illness cannot be eliminated at this time, it must be managed.

- How can the risk of O157 illness best be managed?

A general approach to manage the risk of O157 illness attributable to ground beef is: (1) to reduce the level of O157 on the farm, and (2) to better understand different channels of the ground beef production system and use this knowledge to identify critical points at which intervention would be most effective. To gain a better understanding of the system, specific questions that need to be addressed include: (a) how does the number of steps involved in the production of ground beef affect the risk of contamination?, (b) how does the risk change as ground beef moves through the system?, and (c) what is the volume of ground beef that flows through the various channels? If it is possible to identify one or two points along the continuum that can be associated with an increased risk of O157 contamination, then research can be focused on those specific channels.

- Where should attention be focused?

Attention should be focused on what occurs just prior to slaughter. Because shedding of O157 may be sporadic, cattle that test O157-negative on the farm may test positive just prior to slaughter. This is especially plausible in light of the many stress factors to which cattle are subjected between leaving the farm or feedlot and slaughter. Although it is not known if cattle that are not shedding O157 at the time of slaughter can be a source for ground beef contamination, animals which are shedding can be a factor in such contamination. Thus, individual cattle should be followed and sampled at various points after leaving the farm. Sampling at the auction barn, feedlot, after unloading at the slaughter plant, and immediately before slaughter may provide valuable information about shedding patterns. The cleanliness of animals entering the slaughter facility is also an important consideration. Contamination of the hide and haircoat with mud and feces may provide O157 with an additional mode of entry into the slaughter facility via either culture-positive or culture-negative animals.

- What other types of preharvest research should be recommended?

Research should concentrate on the ecology of O157 in the gastrointestinal tract of ruminants, specifically to assess the effects of stressors such as dietary changes and movement of animals. The ecology of O157 in the farm environment also needs further research. Since previous studies of management factors, such as the use of ionophores, have not been definitive, further work is needed to address the effects of management factors on the prevalence of O157. Competitive exclusion, the administration of protective intestinal microorganisms known as probiotics, should also be evaluated as an intervention strategy. Probiotics can protect poultry from colonization by human enteropathogens, including O157. Results of studies on the use of probiotics in cattle have been variable. None of the currently available probiotic feed supplements for cattle marketed in the U.S. has met the regulatory requirements for demonstration of prophylactic or therapeutic claims.

- What about postharvest research?

Emphasis should be placed on identifying and monitoring where and how contamination occurs. The Hazard Analysis and Critical Control Point (HACCP) system should continue to be developed and implemented as a preventative food safety assurance system. HACCP principles should be applied not only at slaughter and grinding facilities but also at other points along the continuum including shipment between locations and storage. The intent would be to ensure that a product leaving a certain phase of production or location is as safe or safer than when it entered.

- What about tracebacks?

Tracebacks have been proposed as an important component of a food safety agenda. In the case of O157, tracebacks could provide valuable information about on-farm factors and production processes associated with the organism, as well as about the ecology of O157. However, from an immediate disease prevention perspective, tracebacks would currently be of uncertain value. Not enough is known about the ecology of O157 in cattle to implement prudent, on-farm measures to prevent future contamination. Tracebacks involving ground beef would be especially difficult to carry out to the farm level with a high degree of precision. Even given a highly dependable system of individual animal identification, the complexity of production and distribution channels for ground beef tends to make the determination of individual animal contributions to any given pound of product a difficult process.

Table S.1. Association of *E. coli* O157:H7 with Selected Management Practices[*]

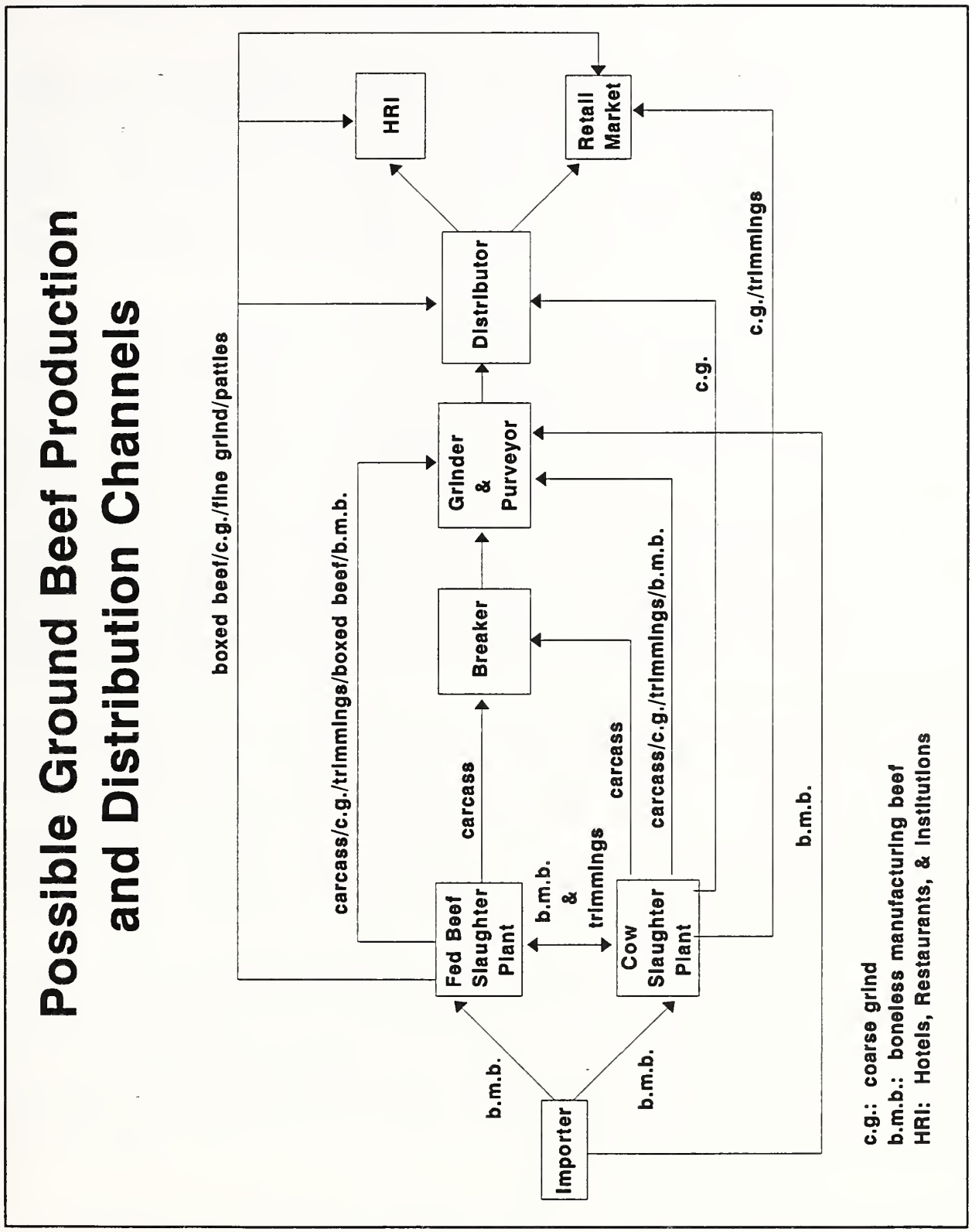
Management Practice	Subgroup	Association with O157
Small herd size[a,b]	Dairy farms	pos, none
Use of computerized feeders[a]	Dairy farms	pos
Irrigation of pastures with manure slurry[a]	Dairy farms	pos
Feeding of whole cottonseed[a,c]	Dairy heifers and cows	neg, neg
Feeding of milk replacer[b,c]	Dairy calves	neg, none
Feeding of ionophores[b,c]	Dairy calves	pos, none
Grouping of calves prior to weaning[b,c]	Dairy calves	none, pos
Sharing of unwashed feeding utensils among calves[c]	Dairy calves	pos
Feeding of oats in starter ration[c]	Dairy calves	pos
Feeding of grain during first week of life[b,c]	Dairy calves	none, pos
Feeding of clover as first forage[c]	Dairy calves	neg

[*] Many other management factors have been tested for association with O157; only those listed were found to have statistical significance at $p \leq 0.10$.

pos = positive association, i.e., management practice is associated with increased O157 prevalence
neg = negative association, i.e., management practice is associated with decreased O157 prevalence
none = no association

[a] Hancock et al., 1994
[b] Hancock et al., 1993b
[c] Garber et al., 1994

Figure S.1


Possible Ground Beef Production and Distribution Channels
HRI
Retail Market
Distributor
Grinder & Purveyor
Breaker
Fed Beef Slaughter Plant
Cow Slaughter Plant
Importer
boxed beef/c.g./fine grind/patties
carcass/c.g./trimmings/boxed beef/b.m.b.
carcass
carcass
b.m.b. & trimmings
carcass/c.g./trimmings/b.m.b.
c.g.
c.g./trimmings
b.m.b.
b.m.b.
b.m.b.
c.g.: coarse grind
b.m.b.: boneless manufacturing beef
HRI: Hotels, Restaurants, & Institutions

Introduction

Although *Escherichia coli* O157:H7 (O157) was first recognized as a human pathogen in 1982, only recently has O157 emerged as a well-known cause of foodborne illness in the U.S. O157 results in far fewer human illnesses than other foodborne pathogens, such as *Salmonella*, but those who become infected with O157 have a greater chance of suffering severe illness or death. Consumers began to question the safety of the nation's food supply when over 600 individuals became ill and 4 children died in an outbreak which began in Washington state in late 1992 after a fast food restaurant chain served undercooked hamburgers contaminated with O157. The occurrence of additional outbreaks and deaths in 1993 prompted investigation into why more illness due to O157 is being seen and what can be done to prevent future human illness.

An increase in the number of human cases reported to be associated with this organism indicates that the prevalence of the organism has increased, that human exposure to O157 has increased, and/or that recognition and reporting of the illnesses has increased. Because many human outbreaks have been associated with the consumption of bovine-origin products, and because some O157 strains found in cattle are identical to those associated with human disease, there is speculation that cattle are the primary source for human cases of O157. Further, some theorize that the prevalence of O157 among cattle is increasing. The continuing occurrence of human cases does indicate that O157 is being maintained in some type of reservoir.

Although consumers can take measures to protect themselves from O157 and other foodborne pathogens through proper food handling and cooking, much attention has focused on preharvest control measures to further reduce the potential for human disease. In order to address the issues surrounding preharvest control of O157, it is necessary to understand the ecology of the organism, including reservoirs and modes of transmission to humans. Because ground beef has been implicated as a major source of O157 disease outbreaks, it is also important to understand the farm-to-consumer continuum for ground beef.

Information used in this report came from various sources. An extensive literature search provided much of the background. Analyses are based in part on data from the National Agricultural Statistics Service (NASS), Food Safety and Inspection Service (FSIS), Packers and Stockyards Administration (P&SA), Human Nutrition Information Service (HNIS), Animal and Plant Health Inspection Service (APHIS), Economic Research Service (ERS), and Agricultural Marketing Service (AMS) -- all agencies of the U.S. Department of Agriculture (USDA). Additional information came from personal communications with representatives of the cattle industry and other experts. A survey of packers, conducted in conjunction with the American Meat Institute (AMI), contributed information that was unavailable elsewhere.

This document is macro-epidemiologic in scope. It presents what is currently known about O157, explores potential avenues to increase our understanding of this pathogen, and addresses potential intervention strategies with emphasis on the preharvest level. The document is divided into six sections: 1) the biologic properties of O157 and the effect of environmental conditions on the organism; 2) O157-associated disease in humans; 3) O157 in cattle and risk factors associated with its presence; 4) foods, including raw milk and ground beef, as vehicles for transmission of O157 to humans; 5) the ground beef continuum from farm to consumer; and 6) intervention, prevention, and control of O157.

The Organism: *Escherichia coli* O157:H7

How is O157 different from other *Escherichia coli*?

Hundreds of types of *Escherichia coli* (*E. coli*) exist, many of which are normal inhabitants of the lower intestines of warm-blooded animals. Carnivores and omnivores usually harbor the organisms in greater numbers than herbivores. The feces of cows and horses normally contain 10^3 to 10^4 *E. coli* per gram (Timoney et al., 1988).

E. coli are gram-negative, rod-shaped bacteria that vary in morphology under different conditions. They can be nonmotile or motile by means of flagella. *E. coli* never form spores and stain readily and evenly with ordinary stains. *E. coli* grow readily on ordinary media at an optimum temperature of 37°C and are aerobic and facultatively anaerobic (can survive in low to no oxygen environments) (Timoney et al., 1988).

The immunologic properties of the surface structures of *E. coli* are described as O (somatic), K (capsular), H (flagellar), and F (fimbria) antigens (Timoney et al., 1988). At least 170 O, 80 K, and 56 H antigens are recognized. Each serotype is designated by the numbers of the antigens it carries. *Escherichia coli* O157:H7 (O157) is identified by its O (157) and H (7) antigens.

One way O157 differs from other *E. coli* serotypes is that it ferments sorbitol very slowly and does not produce β-glucuronidase. These characteristics are used to differentiate O157 from other *E. coli* serotypes on selective media such as sorbitol MacConkey agar and/or 4-methylumbelliferyl-β-D-glucuronide (MUG) (Kim and Doyle, 1992; Pawelzik, 1991). Several laboratory procedures exist to identify and specifically isolate O157 once bacterial colonies are plated; however, there is no single standardized method for O157 identification (Khakhria et al., 1990; Perry et al., 1988; Borczyk et al., 1990; Ronner and Cliver, 1990; Okrend et al., 1990; Szabo et al., 1990; Chapman et al., 1991; Dorn and Angrick, 1991; Farmer et al., 1988; Speirs and Stavric, 1989).

E. coli can produce several toxins that act on the intestinal tract and other organs of humans and animals. These toxins can produce disease and have been classified into four groups: enterotoxigenic *E. coli* (ETEC), enteroinvasive *E. coli* (EIEC), enteropathogenic *E. coli* (EPEC), and enterohemorrhagic *E. coli* (EHEC) (O'Brien and Holmes, 1987). O157 is classified as an EHEC. EHEC produce Shiga-like toxins (verotoxins) which are similar to toxins produced by *Shigella dysenteriae*.

How does O157 compare to other foodborne pathogens?

Salmonella, like *E. coli*, are gram-negative organisms that inhabit the lower gastrointestinal tracts of mammals. O157 and most *Salmonella* have flagella and frequently also carry fimbriae. Both O157 and *Salmonella* contain O and H antigens; in fact, O157 possesses a somatic (O) antigen identical to that of *Salmonella* $O30_1$ (Shimada et al., 1992). Some *Salmonella* strains, such as *Salmonella typhimurium*, produce toxins including enterotoxin and verotoxin (O'Brien et al., 1982; Strockbine et al., 1985). Other foodborne pathogens that produce toxins which can be neutralized by anti-Shiga toxin include some strains of *Shigella* spp., *Vibrio* spp., and *Campylobacter jejuni* (O'Brien et al., 1984). *S. typhimurium*, *C. jejuni*, and O157 share similar temperature requirements and all are capable of survival and growth in low oxygen environments (Table 1.1).

How do environmental conditions affect O157?

Little is known about the conditions conducive to survival and growth of O157 in farm or feedlot environments. Data from laboratory isolation of the organism and pathogenesis studies provide information on differences between O157 and other *E. coli* organisms and give clues to what may be required for survival of the organism in the environment. Given the similarities between O157 and some other foodborne pathogens noted previously, it is reasonable to assume that O157 may behave much like those organisms under similar environmental conditions.

In some respects, however, O157 is actually hardier than some other foodborne pathogens. *S. typhimurium* and *C. jejuni* do not appear to be as tolerant of acidic conditions (low pH) as O157, which can survive in processed meat products at a pH as low as 4.5 (Doyle and Schoeni, 1984; Glass et al., 1992). O157 can survive in ground beef frozen at -20°C (-4°F) for up to 9 months, but is susceptible to cooking temperatures in excess of 60°C (140°F). Thus, O157 is at least as heat sensitive as some other common foodborne pathogens (Tables 1.1 and 1.2).

Cases of O157 have resulted from consuming raw milk and unchlorinated water, indicating that the organism can survive in those environments (Herriott, 1993; Swerdlow et al., 1992). Pasteurization of milk at 60°C (140°F) to 72°C (162°F) for a minimum holding time of 16.2 seconds inactivates O157 (D'Aoust et al., 1988). Laboratory studies have shown that O157 can survive for more than 70 days in water at 5°C (41°F) and for up to 40 days at 20°C (68°F) (Rice et al., 1992).

O157 can survive for some time in manure/soil as evidenced by a human case involving a lacto-ovo-vegetarian (Cieslak et al., 1993). The affected person ate vegetables from a garden fertilized with cow manure. Based on this survival in manure/soil and an apparent resistance to freezing, O157 may be able to overwinter in the farm environment.

Table 1.1. Characteristics of Three Selected Foodborne Pathogens

Characteristic	*E. coli* O157:H7	*Salmonella typhimurium*	*Campylobacter jejuni*
pH tolerance	4.5-9.0	6.2-9.0	6.0-9.0
Growth temperature and survivability	Poor at 44-45°C; no growth at 45.5°C. Can survive 9 months at -20°C in ground beef. Can survive >70 days in 5°C water.	6.2-45°C; best at 35-37°C.	Best at 42°C; no growth at 25°C. At -18°C in ground beef, 10^5 reduced by 1 log over 7 days.
Toxins	Verotoxin	Verotoxin, enterotoxin	Enterotoxin
Oxygen requirements	Facultative anaerobe	Facultative anaerobe	Microaerophilic to anaerobic
Heat sensitivity	D60°C* = 45 seconds in ground beef	Killed at 65°C	D70°C* = 10 minutes in ground beef; D55°C* = 2.25 minutes in ground chicken
Invasiveness	No	Yes	Yes
Antigens	O, H	O, H	O

* D-value is the temperature and time required to destroy 90% of the organisms present
Sources: Griffin and Tauxe,1991; Timoney et al.,1988; Jay,1986

Table 1.2. Time to Death for *E. coli* O157:H7 in Beef Emulsions (30.5% Fat)

Temperature °C	Temperature °F	Time (minutes:seconds)
60.0	140	8:20
62.2	145	2:07
65.6	150	0:32
68.3	155	0:08

Source: Line et al.,1991

E. coli O157:H7 in Humans

How did O157 become a human pathogen?

Several hypotheses address the relatively recent emergence of O157 as a human pathogen. One hypothesis is that O157 developed from another *E. coli* fairly recently (Whittam et al., 1993). Another is that the organism emerged long ago, but only recently acquired the ability to produce verotoxin through a bacteriophage vector. A third hypothesis is that O157 has been present for a long period of time, but that changes in management practices associated with cattle production have favored its presence in recent years (Hancock et al., 1993a). Regardless of how or why O157 emerged, it is very clear that O157 is now a significant foodborne pathogen that may cause serious illness in humans.

How has O157 been transmitted to humans?

Transmission of O157 to humans may occur directly or indirectly. Direct O157 transmission from cattle, probably through oral contact with feces, occurred on one premises where children interacted with calves (Renwick et al., 1993). Person-to-person transmission by fecal-oral contact is typical of settings such as day care centers (Belongia et al., 1993; Ratnam et al., 1986; Spika et al., 1986). Indirect transmission, such as through O157-contaminated foods, has occurred frequently. Food may be contaminated by contact with feces containing O157, or cross-contaminated by O157-contaminated soil, food, preparation surfaces, utensils, etc. (Cieslak et al., 1993). O157 has also been transmitted to humans through contaminated water (Swerdlow et al., 1992; McGowan et al., 1989; Dev et al., 1991).

What is the pathogenesis of O157 in humans?

Adherence to intestinal mucosal cells and production of Shiga-like toxin are thought to be the major virulence properties of O157 (Griffin and Tauxe, 1991). O157 is not invasive in standard *in vitro* assays but adheres to mucosal cells of the intestine and destroys the microvilli (O'Brien and Holmes, 1987). The major sequelae to O157 infection -- hemorrhagic colitis (HC), hemolytic uremic syndrome (HUS), and/or thrombotic thrombocytopenic purpura (TTP) -- appear to be caused by the verotoxin, although the specific mechanisms have not yet been clearly defined. HC may be produced when O157 verotoxin is released into the lumen of the colon where it kills surface epithelial cells by inhibiting protein synthesis (O'Brien and Holmes, 1987). HUS or TTP may result when verotoxin enters the bloodstream and binds to receptors on vascular endothelial cells in the kidneys (O'Brien and Holmes, 1987).

The infectious dose of O157 has not been determined, but is believed to be low in comparison with other pathogenic foodborne bacteria (Table 2.1). Sample cultures taken from raw ground beef implicated in a U.S. outbreak in 1993 recovered as few as 15 organisms per gram (Personal communication, J. Hollingsworth, USDA:FSIS, November 1993). Frozen ground beef associated with a Canadian outbreak was found to have 100 organisms per gram (Todd et al., 1988).

Following a 3- to 12-day incubation period (Ryan et al., 1986), patients infected with O157 typically present with severe abdominal cramps and bloody diarrhea, but rarely have a fever (Griffin and Tauxe, 1991; Padhye and Doyle, 1992). An estimated 20 to 30 percent of O157 cases present with nonbloody diarrhea (Harris, 1990). Consequently, O157 may be misdiagnosed as inflammatory bowel disease, ischemic colitis, intussusception, or obstructive bowel disease (Griffin et al., 1990).

In most patients, O157 illness is self-limiting. Treatment consists of supportive care and monitoring for potential complications such as HUS and TTP (Padhye and Doyle, 1992). HUS consists of hemolytic anemia, thrombocytopenia, and renal failure. Children are at highest risk of developing HUS, which is thought to be a leading cause of acute kidney failure in the young. HUS develops in 5 to 10 percent of persons with O157 and 5 percent of HUS patients die (Neill, 1989; Speirs and Stavric, 1989; Crosse and Naylor, 1990). TTP, which is more common in adults, is an extension of HUS and is distinguished by its more frequent and severe neurologic involvement.

Infected humans shed O157 in the feces for an average of 17 days (range: 2 to 62 days). Some patients may shed intermittently and shedding duration is unrelated to bloody versus nonbloody diarrhea or development of HUS (Belongia et al., 1993).

Although subclinical cases are a potential source of infection in the community, healthy human carriers are believed to be rare and are probably not an important reservoir (Personal communication, P. Tarr, Children's Hospital, University of Washington, November 1993). However, large scale fecal cultures from asymptomatic individuals have not been performed.

O157 may go undetected if fecal culture is not requested by the patient's physician or initiated by the laboratory, or if sorbitol MacConkey agar is not used. The ability to recover O157 is markedly reduced if a fecal culture is not performed within 6 days after the onset of illness (Remis et al., 1984; Christie et al., 1990; Swerdlow et al., 1992). Although O157 is most commonly diagnosed through fecal culture, the organism has rarely been isolated from extraintestinal sites such as human urine, the glans penis, and blood (Griffin et al., 1988).

Is there any surveillance for human cases of O157?

The Centers for Disease Control and Prevention (CDC) receives abstracted reports from states on O157 cases, and maintains a national data base of O157 investigations. In most states, O157 surveillance is based on laboratory reporting; however, clinicians and/or hospitals may share the reporting responsibility. An underreporting of O157 cases is likely since most states do not require that their health departments be notified of suspected cases and because culturing for O157 may not be routinely performed. As of the end of 1993, reporting of O157 cases to the state health department was required in only 18 states: Connecticut, Idaho, Iowa, Michigan, Minnesota, Mississippi, Missouri, Nebraska, Nevada, New Jersey, New Mexico, New York, North Dakota, Rhode Island, South Carolina, Ohio, Oregon, and Washington. Eight more states plan to require case reporting beginning in 1994 (Personal communication, W. Keene, Oregon Dept. of Health, November 1993). Washington was the first state to require reporting, in 1987.

What is the human incidence of O157 in the U.S.?

The CDC estimates that from 10,000 to 20,000 cases of O157 infection occur in the U.S. each year (CDC, 1993a). Based on studies conducted in Washington state, the O157 incidence rate in the U.S. population is estimated at about 8 per 100,000 persons per year (Dorn, 1988; MacDonald et al., 1988; Griffin and Tauxe, 1993). Higher rates have been found in individuals under 5 years and those 65 years of age and older (Pai et al., 1988). Although relatively low in incidence as compared to other foodborne pathogens, O157 appears to be widespread with human cases reported from at least 20 states.

Evidence indicates that the incidence of O157 in the U.S. is increasing and that the increase is not solely a reporting artifact. The CDC has no record of outbreaks of bloody diarrhea of unknown origin before 1982, suggesting that O157 was not likely to have been a frequent cause of outbreaks

before that time (Griffin and Tauxe, 1991). Also, a retrospective review of over 3,000 *E. coli* serotypes identified by the CDC from 1973 through 1983 found only one O157 -- a 1975 isolate associated with a case of bloody diarrhea in a 50-year-old California woman (Riley et al., 1983).

Because O157 is the predominant enteric pathogen in sporadic HUS following diarrhea, HUS can serve as a sentinel for O157 (Neill, 1989). One study reported isolating O157 from over 63 percent of HUS patients (Table 2.2). In Minnesota, the incidence of HUS in children less than 18 years old increased from 0.5 to 2.0 per 100,000 between 1979 and 1988 (Martin et al., 1990). A study in the state of Washington indicated that between 1976 and 1980, HUS incidence among children under age 15 was about 2.5 times higher than between 1971 and 1976 (Tarr et al., 1989).

The true human incidence or prevalence of O157 can only be estimated. The presence of bloody diarrhea commonly warrants visiting a physician, but infected persons showing no symptoms and persons with nonbloody diarrhea are less likely to seek medical attention. Thus, most studies of O157 are likely to underestimate the incidence or prevalence of human illness.

How many outbreaks and clusters of O157 have been reported in the U.S.?

Through 1993, 32 outbreaks[1] of O157 were documented in the U.S. (Tables 2.3 and 2.4). Thirteen (41%) of those occurred in 1993. Since 92 percent of the 1993 outbreaks involved just 11 or fewer confirmed cases, the large number of outbreaks in 1993 could have been due in part to increased awareness and reporting. At the end of 1993, 18 states required reporting of O157 cases (Personal communication, W. Keene, Oregon Dept. of Human Resources, November 1993).

Twelve (38%) of the outbreaks were associated with some type of ground beef. Other vehicles identified were roast beef, raw milk, apple cider, hot dogs, and unchlorinated water. Although the most widely publicized outbreaks have involved restaurants, only 7 (22%) of the 32 total outbreaks reported in the U.S. have been associated with such settings.

In addition to the outbreaks, several clusters[2] of O157 cases were documented in 1993 (Table 2.5). Four cases in Maine could be linked to a single slaughter plant, but several unrelated cases occurred throughout the state between June and August (*Federal Veterinarian*, 1993). In October and November, 10 cases of O157 were reported in Texas, none of which were related. Eight of the cases developed HUS, and two individuals died (Personal communications: T. Gomez, CDC, Atlanta, GA, December 1993; J. Harman, Texas Dept. of Health, Austin, TX, January 1994).

What are the risk factors for human infection?

Several factors may be associated with increased risk of human O157 infection. The consumption of bovine products is currently the most important risk factor. Age and institutional settings may be significant, but the significance of other factors is not yet well understood.

Bovine product consumption. Of the 24 U.S. outbreaks in which a food vehicle was identified, 17 (71%) involved the consumption of a bovine-origin product (Tables 2.3 and 2.4). Fifteen were

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[1] An O157 outbreak is defined as an incident in which two or more persons experienced similar illness and epidemiologic analysis implicated a common exposure.

[2] A cluster of O157 cases are those which were suspected of having been related to a common exposure, even though no specific vehicle could be identified epidemiologically.

associated with some type of beef and two with raw milk. In each of the seven outbreaks where a nonbovine origin food item was implicated as a vehicle, cross-contamination from a bovine product was suspected.

Age. As is the case with several other foodborne pathogens, the very young and the elderly are at the greatest risk for O157 illness. The age-specific prevalence is highest in children under 5 years of age, gradually decreases with age, then increases again in persons age 65 or greater (Pai et al., 1988). The very young and very old are most susceptible to O157 sequelae (e.g., HUS and TTP) and death from disease-associated complications.

Institutional settings. About 30 percent (10/32) of U.S. outbreaks have been related to day care centers, schools, mental health facilities, or nursing homes. People associated with such institutions are likely to have similar dietary intakes; therefore, if O157 is present in a food item, a potentially large number of people may be exposed. Age is a likely correlate to institutions, since day care centers and nursing homes house higher risk individuals.

Person-to-person transmission in day care centers poses a particular risk to young children. In such settings, O157 may be readily transmitted from person-to-person through improper diaper changing practices, inadequate toilet hygiene, and failure to exclude ill children. In a Minnesota study of nine day care centers in which at least one O157-infected child was present, the O157 infection rates of attending children ranged from 3 to 38 percent (median=22%) (Belongia et al., 1993).

Season. Both outbreaks and sporadic cases of O157 peak during the warmest months of the year. Several studies of sporadic cases in the U.S. reported a higher incidence or prevalence of O157 from May through September (Table 2.2). In addition, at least three O157 outbreaks have been observed in each of the months from May through November, with 28 (88%) of the 32 U.S. outbreaks having occurred in one of those 7 months (Figure 2.1). The reasons for the seasonal pattern of cases and outbreaks is not known, but they could hypothetically include: 1) an increased prevalence of O157 in cattle or other livestock during the summer; 2) greater human exposure to ground beef or other O157-contaminated foods during the "cook-out" months; and/or 3) more improper handling (temperature abuse) or incomplete cooking of products such as ground beef during warmer months.

Geographic region. Regional prevalences are difficult to compare because of differences in recognition, testing, and reporting of O157. A total of 28 (88%) of the U.S. outbreaks were reported from northern or western states. Twenty-four (75%) were in northern states and 19 (59%) in western states (15 outbreaks were in states considered to be both northern and western). Although such a majority of documented outbreaks in the North and West could be due to a reporting phenomenon, a recent 2-year hospital study did find a significantly greater number of stool specimens positive for O157 in northern and western states than in southern states ($p<0.01$) (Unpublished data, P. Griffin, CDC, August 1993). CDC surveillance in 28 states from 1982 to 1984 found O157 infections distributed throughout the country with no apparent geographic clustering (Remis et al., 1984).

Occupation. A higher occupational risk may exist for people exposed to cattle, contaminated ground beef, or clinical stool specimens, but the risk has not been well quantified (Ostroff et al., 1989; Griffin and Tauxe, 1991). In 1992, evidence for calf-to-human transmission was provided by the isolation of the identical phage type of O157 from calves and a toddler in Ontario, Canada (Renwick et al., 1993). The mother of the infected child noted that the boy touched the calves and put his fingers into both the mouths of the calves and into his own.

Alteration of the gastrointestinal tract or its microflora. A study of a nursing home outbreak in Canada suggests that previous gastrectomy or recent antimicrobial therapy may increase the risk of O157 illness (Carter et al., 1987; Griffin and Tauxe, 1991). Both antibiotics and gastrectomy could allow O157 to more readily colonize the human intestinal tract. In addition, although most O157 are not resistant to antibiotics, antibiotic treatment after the onset of diarrhea may be associated with development of HUS (Griffin and Tauxe, 1991). One possible explanation for this finding is that severely ill patients who are more likely to develop HUS may also be more likely to receive antibiotics.

What is the cost of illness due to O157 in the U.S.?

The USDA's Economic Research Service has ranked O157 as the fourth most expensive foodborne pathogen, after *Toxoplasma*, *Salmonella*, and *Campylobacter*. Yearly medical costs and productivity losses attributed to O157 have been estimated at $229 to 610 million, largely due to deaths of young persons from kidney failure (USDA:ERS, 1993).

What about human cases of O157 in other countries?

In addition to the U.S., at least 14 countries have documented the presence of O157 in human populations (Table 2.6). Three additional countries found *E. coli* O157, but have not confirmed the presence of O157:H7 (Lerman et al., 1992; Cordovez et al., 1992; Chart et al., 1991). The organism has not been detected in the human populations of New Zealand, Thailand, Korea, India, and Brazil (Personal communication, M. Muirhead, NZ Meat Products Board, July 1993; Bettelheim et al., 1990; Kim et al., 1989; Bhan et al., 1989; Giraldi et al., 1990). For many countries, no published reports could be found on the status of O157.

Countries which have documented the presence of O157 are located on six different continents, although to date O157 has been reported mostly from nontropical regions (Figure 2.2). The only reported finding of O157 within the tropics has been from a rural village in southwestern Mexico in the mid-1980's (Cravioto et al., 1990). The lack of detection of the organism in tropical regions of the world may be a reporting artifact, as many subtropical countries are less well-developed in terms of disease detection and reporting infrastructures. The finding of O157 in rural Mexico just a few years after the first known outbreaks in the U.S. may be evidence that this organism existed for quite some time prior to its recognition as a human pathogen.

Canada has the highest rates of isolation of O157 from a widely sampled human population ($\geq$1,000 samples) (Table 2.7). In Alberta, 2.5 percent (137/5,415) of patients that had a stool sample submitted to any of 3 hospital laboratories were found positive. In Newfoundland and Labrador, 1.7 percent (18/1,043) of stools submitted to a public health laboratory were positive for O157 (Pai et al., 1988; March and Ratnam, 1986).

Most human cases of O157 outside the U.S. have been sporadic in nature. The only known outbreaks outside the U.S. have occurred in Canada and the United Kingdom (UK). Ground beef has been implicated as a vehicle in at least two outbreaks in Canada (Hockin et al., 1987; Hockin and Lior, 1987).

The number of human cases of O157 in Canada, the UK, and Belgium appears to peak during the warmest months of the year (Table 2.7). In Alberta, Canada, in 1985, the greatest number of cases occurred in July and August (Pai et al., 1988). Cases of HC associated with O157 in England were found at a higher rate in the summer and cases of HUS peaked in July and August in both Canada

and the UK (Chapman et al., 1989; Hall, 1985; PHLS Communicable Disease Surveillance Center, 1990; Rowe et al., 1991).

Table 2.1. Characteristics of Human Disease Caused by Three Selected Foodborne Pathogens

Characteristic	*E. coli* O157:H7	*Salmonella typhimurium*	*Campylobacter jejuni*
Human infectious dose (cells / gram)	Believed to be $\leq 10^2$	10^7-10^9	10^3-10^6
Incubation period	4-8 days	8-14 hours	24-82 hours
Human symptoms	Vomiting, abdominal cramping, watery then bloody diarrhea, no fever; may require hospitalization; can progress to HUS/TTP	Vomiting, abdominal pain, headache, chills, diarrhea, prostration, moderate fever	Abdominal pain or cramping, diarrhea, malaise, headache, fever; diarrhea may resemble ulcerative colitis in severe cases
Mortality rate	≤2%	≤0.1%	≤0.01%
Shedding and distribution	Humans can shed ≥1 month after diarrhea onset; cattle being investigated as carriers; can colonize chicken ceca; other animals not been adequately tested	Up to 5% of humans shed upon recovery; found in birds, reptiles, farm animals	Up to 8% of humans may be chronic shedders; may be most common foodborne cause of human diarrhea; found in poultry and all major meat animals
Site of human pathology	Colon	Small intestine	Small intestine
Estimated no. of annual cases	10,000-20,000	2 million	1 million
Most common food vehicle(s)	Ground beef (bovine products)	Beef, poultry, homemade ice cream, pork	Poultry, raw milk

HUS = hemolytic uremic syndrome; TTP = thrombotic thrombocytopenic purpura
Sources: CDC,1993a; Griffin and Tauxe,1991; Timoney et al.,1988; Jay,1986; USDA:ERS,1993

Table 2.2. Studies of Human *E. coli* O157:H7 in the U.S.

Location	Period	Prevalence / Incidence	Comments	Reference
U.S. (28 states)	1982 - 1984	37% of patients (28/76) with HC	CDC surveillance; positives were in 11 states	Remis et al., 1984
WA (health maintenance organization)	1985 - 1986	7.6 per 100,000 persons/year; 0.4% of stools positive; 0.55% of patients positive	Prevalence similar to *Campylobacter* and greater than *Shigella* or *Salmonella*; 76% from May-Aug.	Dorn,1988; MacDonald et al.,1988
WA	1985 - 1986	100% (7/7) of HUS patients with HC	Half of siblings had nonbloody diarrhea	Neill et al., 1987
WA	1985 - 1987	63.5% of all HUS cases positive; 67.3% of HUS patients with HC	Included 12 patients from study by Neill et al.,1987	Christie et al., 1990
PA (hospital)	1986	0.08% (3/3,710) of stools from children	1 of 15 water samples (streams, reservoirs) positive	McGowan et al., 1989
MN (Mayo Clinic)	1987	0.5% of submissions (10/2,164) positive	Fourth most common bacterial stool pathogen	Marshall et al., 1990
WA	1987	2.1 per 100,000 persons/year	67% from May-Sep.	Ostroff et al., 1989
U.S. (10 hospitals)	1991 - 1992	0.38% of all stools (99/26,239) positive	More positives in north & west ($p<0.01$); 64% from June-Sep.	*

HC = hemorrhagic colitis; HUS = hemolytic uremic syndrome

* Unpublished data, P. Griffin, CDC, August 1993

Table 2.3. Reported Outbreaks of *E. coli* O157:H7 in the U.S., 1982-1992.

No.	State(s)	Onset	Setting	Source(s)	Comments	Reference(s)
1.	OR	Feb. 1982	Fast food	Ground beef (hamburger)	26 ill	Riley et al.,1983
2.	MI	May 1982	Fast food	Ground beef (hamburger)	21 ill; related to Oregon outbreak	Riley et al.,1983
3.	NC	Sep. 1984	Day care	Person-to-person	36 ill, 3 HUS	Spika et al.,1986
4.	NE	Sep. 1984	Nursing home	Ground beef (hamburger)	34 ill, 1 HUS, 4 died	Ryan et al.,1986
5.	NC	May 1986	Day care	Person-to-person	15 ill, 2 HUS	*
6.	WA	Oct. 1986	Community	Ground beef; ranch dressing	37 ill, 4 HUS, 2 died	Ostroff et al., 1990; Griffin et al.,1988
7.	UT	June 1987	Mental institution	Ground beef; person-to-person	51 ill, 8 HUS, 4 died	Pavia et al.,1990
8.	WI	May 1988	School	Roast beef	61 ill	Griffin and Tauxe,1991
9.	MN	Aug. 1988	Day care	Person-to-person	19 ill, 3 HUS	Belongia et al., 1993
10.	MN	Oct. 1988	School	Ground beef (pre-cooked patties)	54 ill children	Belongia et al., 1991
11.	MO	Dec. 1989	Community	Unchlorinated water	243 ill, 2 HUS, 4 died; under-diagnosed due to failure to culture	Swerdlow et al., 1992; Rice et al.,1992
12.	ND	July 1990	Community	Roast beef	65 ill, 2 HUS	Griffin and Tauxe,1991
13.	MT	Nov. 1990	School	School lunch	10 ill, 1 HUS	Griffin and Tauxe,1991
14.	OR	July 1991	Community	Swimming water	21 ill, 4 HUS; swam in lake	*
15.	MA	Nov. 1991	Community	Apple cider	23 ill, 3 HUS	*
16.	NV	June 1992	Day care	Person-to-person	57 ill	*
17.	ME	Sep. 1992	Home	Vegetables; person-to-person	4 confirmed cases, 1 HUS, 1 died; vegetables grown in manure-fertilized garden	Cieslak et al., 1993
18.	WA (CA, NV, ID)	Nov. 1992	Fast food	Ground beef (hamburger; person-to-person)	607 ill, 56 HUS, 4 died	CDC,1993b; Dorn,1993; *
19.	OR	Dec. 1992	Community	Raw milk	9 confirmed cases; 6 drank raw milk traced to 1 farm	Herriott,1993

HUS = hemolytic uremic syndrome

* Personal communication, T. Gomez, CDC, Atlanta, GA, 1994

Table 2.4. Reported Outbreaks of *E. coli* O157:H7 in the U.S., 1993.

No.	State(s)	Onset	Setting	Source(s)	Comments	Ref.
1.	OR	March	Restaurant (2 locations)	Mayonnaise	19 confirmed cases	*
2.	OR	June	Community	Raw milk	5 confirmed cases, 2 HUS (10 ill)	Herriott, 1993
3.	CA	July	Community	Ground beef (home-cooked)	3 confirmed cases, 8 probable cases, 1 died; 3 of 30 meat samples O157-positive	*
4.	MA	July	Community	Ground beef	5 confirmed cases, 3 HUS (10 ill)	*
5.	NC	July	Day care	Person-to-person	7 confirmed cases, 2 HUS (27 ill)	*
6.	WA	July	Church picnic	Pea salad; possible contamination from beef	10 confirmed cases (16 ill); person who prepared salad slaughters own beef	*
7.	NM	July	Building party	Hot dogs (contained beef)	4 confirmed cases, 1 HUS	*
8.	OR	August	Restaurant	Cantaloupe; other salad bar items	10 confirmed cases, 17 presumed cases	*
9.	PA	August	Community	Ground beef (home-cooked)	3 confirmed cases, 1 HUS	*
10.	WA	August	Restaurant	Salad bar	11 confirmed cases (53 ill)	*
11.	CT	September	Country club barbecue	Ground beef (hamburger)	4 confirmed cases (10-15 ill)	*
12.	MT	September	Community	Ground beef	6 confirmed cases, 1 suspected case	*
13.	ID, WA	October	2 fast food restaurants	Unknown	9 confirmed cases, 1 HUS (1 ill food handler)	*

HUS = hemolytic uremic syndrome

* Personal communication, T. Gomez, CDC, Atlanta, GA, 1994

Table 2.5. Reported Clusters of *E. coli* O157:H7 in the U.S., 1993

No.	State	Onset	Comments
1.	ME	June	12 ill, 4 confirmed cases, 2 HUS, 1 died
2.	IL	July	8 ill, 4 confirmed cases, 7 HUS, 3 died
3.	TX	October	10 ill, 5 confirmed cases, 8 HUS, 2 died
4.	WA	October	9 ill, 1 HUS

HUS = hemolytic uremic syndrome

Sources: Personal communications, T. Gomez, CDC, Atlanta, GA, 1994, and J. Harman, Texas Dept. of Health, Austin, TX, 1994

Table 2.6. Human *E. coli* O157[1] in Other Countries

COUNTRY	Human Cases	Human Isolates	Reference(s)
Argentina	Yes	Yes	Lopez et al.,1989
Australia	Yes	Yes	Pryor et al.,1990
Belgium	Yes	Yes	Pierard et al.,1990
Brazil	No	No	Giraldi et al.,1990
Canada	Yes	Yes	March and Ratnam, 1986; Pai et al.,1988
Chile	Yes (:H?)	Yes (:H?)	Cordovez et al.,1992
China		Yes	Kain et al.,1991
Czechoslovakia		Yes	Sramkova et al.,1990
Germany		Yes	Wuthe, 1987
India	No	No	Bhan et al.,1989
Ireland		Yes	Meyers et al.,1976
Israel	Yes (:NM)	Yes (:NM)	Lerman et al.,1992
Italy	Yes	Yes	Caprioli et al.,1990
Japan	Yes	Yes	Kobayashi and Harada, 1986
Korea	No	No	Kim et al.,1989
Mexico	Yes	Yes	Cravioto et al.,1990
Netherlands	Yes (:H?)	Yes (:H?)	Chart et al.,1991
New Zealand	No	No	*
South Africa	Yes	Yes	Browning et al.,1990
Spain	Yes	Yes	Smith et al.,1987
Thailand	No	No	Bettelheim et al.,1990
United Kingdom	Yes	Yes	Chapman et al.,1989

[1] All :H7, unless otherwise noted

* Personal communication, M. Muirhead, NZ Meat Products Board, 1993

Table 2.7. Sampling for *E. coli* O157:H7 in Other Countries: Human Population

Location (reference)	Period	No. of Samples	No. Positive (%)	Comments
Belgium microbiology lab. (Pierard et al.,1990)	4/87 to 9/88	3,940 patients	7 (0.2)	71% from May-Sep.; all positives ≤2.5 yrs. of age
Newfoundland/Labrador, Canada public health labs. (March and Ratnam,1986)	1983 to 1984 [a]	1,043	18 (1.73)	
Alberta, Canada 3 hospitals (Pai et al.,1988)	7/84 to 6/86	5,415 patients	137 (2.53)	77% from June-Sep.; age-specific rates: $<5 = 40/10^5$/yr. $\geq 65 = 14.5/10^5$/yr.
Ontario, Canada 2 public health labs. (Cahoon and Thompson,1987)	4/86 to 9/86	7,252	49 (0.68)	92% from June-Sep.
Alberta, Canada 3 hospitals (Bryant et al.,1989)	1986 and 1987	266 patients	103 (38.7)	All patients had bloody diarrhea
Canada public health labs. (Hockin et al.,1988)	1987	?	1,342	5.2 cases per 10^5 persons per year
Ontario, Canada 80 dairies (*)	1992 to 1993	336 persons	1 (0.3)	
England public health lab. (Walker et al.,1988)	6/86 to 9/86	1,319	7 [b] (0.5)	
England (Chapman et al.,1989)	1986 to 1987	40 patients	30 [b] (75)	All patients had HC
		8 patients	5 [b] (63)	All patients had HUS

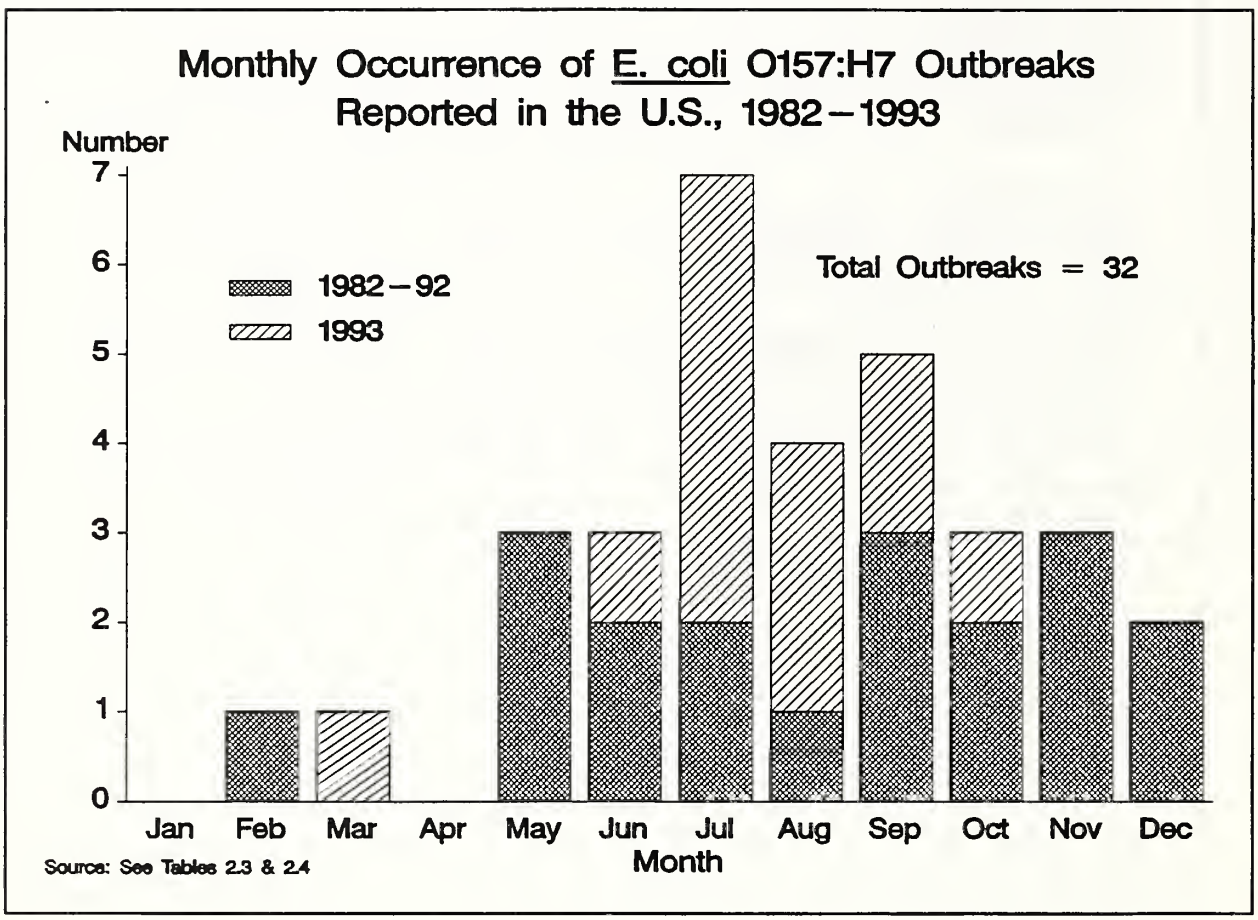
HC = hemorrhagic colitis; HUS = hemolytic uremic syndrome

* Unpublished data, S. Renwick, Agriculture Canada, 1993

[a] Estimated sampling period; actual period not reported

[b] No H-types reported

Figure 2.1


Monthly Occurrence of E. coli O157:H7 Outbreaks Reported in the U.S., 1982–1993
Number
7
6
5
4
3
2
1
0
1982–92
1993
Total Outbreaks = 32
Jan
Feb
Mar
Apr
May
Jun
Jul
Aug
Sep
Oct
Nov
Dec
Month
Source: See Tables 2.3 & 2.4

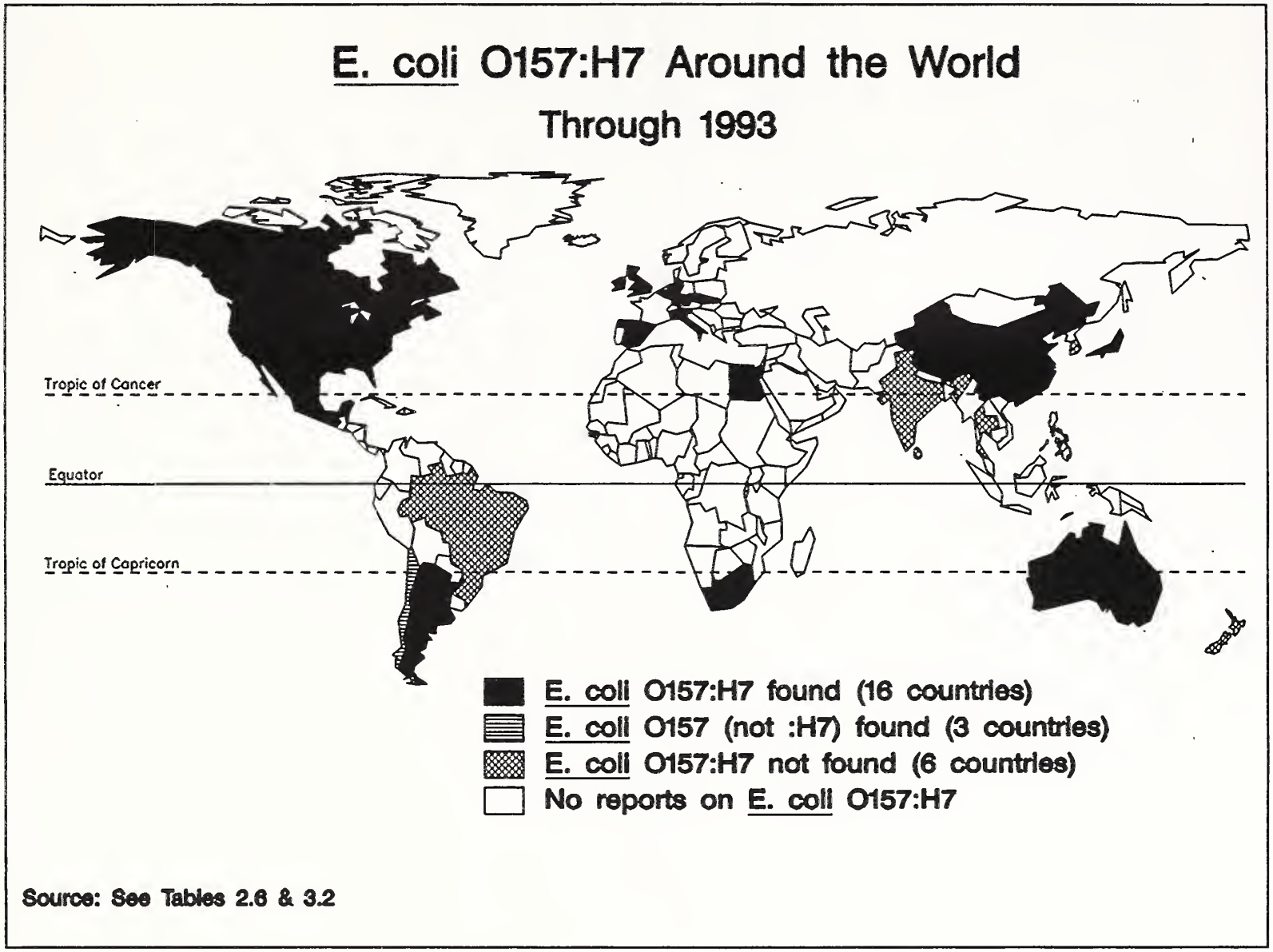

E. coli O157:H7 Around the World
Through 1993
Tropic of Cancer
Equator
Tropic of Capricorn
E. coli O157:H7 found (16 countries)
E. coli O157 (not :H7) found (3 countries)
E. coli O157:H7 not found (6 countries)
No reports on E. coli O157:H7
Source: See Tables 2.6 & 3.2

Figure 2.2

E. coli O157:H7 in Cattle

Why focus on cattle?

The apparent increase in the number of human O157 cases since 1982 raises questions concerning possible reservoirs for the organism and suggests that reservoirs for the agent may have expanded (Hancock, 1993). Bovine products have frequently been associated with human outbreaks of O157 and human disease-associated strains of O157 have been recovered from cattle (Wells et al., 1991; Herriott, 1993; Renwick et al., 1993). It is not absolutely certain, however, that cattle are a reservoir of O157 rather than incidental hosts (Wells et al., 1991; Griffin and Tauxe, 1991; Hancock et al., 1993a). In addition, cattle are not necessarily the only possible source of O157, as very little testing of other animals has been performed in the U.S. Since cattle are known to be associated with O157, this section will focus on cattle and the factors associated with fecal shedding of the organism.

What is the pathogenesis of O157 in cattle?

O157 does not appear to be pathogenic in cattle. The organism is noninvasive and is not known to enter the bloodstream of cattle. Clinical disease associated with O157 in cattle has not been documented in the U.S.

Animal models have been used to study the effects of O157 and its toxins. Numerous studies have revealed that only very young animals with naive gastrointestinal tracts exhibit clinical signs when inoculated with O157 although neither HC nor HUS have been experimentally induced (Farmer et al., 1983; Pai et al., 1986; Beery et al., 1985; Francis et al., 1986; Tzipori et al., 1986). O157 has been recovered from two diarrheic calves outside the U.S., one of unknown age and the other of less than 3 weeks of age (Orskov et al., 1987; Gonzalez and Blanco, 1989).

Unlike other inoculation trials in which older calves have not shown clinical signs, all 4- to 8-week old calves in one study developed diarrhea the day following oral inoculation (Personal communication, W. Cray, NADC, Ames, IA, July 1993). High counts of O157 were found in the colon and cecum of calves necropsied 3 days post-inoculation. The absence of organisms in the liver, spleen, and other organs of the sacrificed calves provides further evidence for the noninvasiveness of O157.

What is the prevalence of O157 in U.S. cattle and cattle herds?

Isolation rates for O157 in the U.S. cattle population have generally been less than one percent, although age-group prevalences of over 5 percent (50 per 1,000) have been found in calves and heifers (Table 3.1). One study reports similar prevalences of about 0.3 percent in feedlot cattle and in dairy cattle (all ages) and a slightly higher prevalence among beef cows (0.7%) (Hancock et al., 1994). This may indicate that dairy cattle are not necessarily the primary source of O157 as some have previously suggested (Wells et al., 1991).

The prevalence of herds with O157 (herd prevalence) has ranged from 1.8 to 100 percent (Table 3.1). Some high herd prevalences may be attributable to the fact that some premises were chosen for sampling because there was evidence that they were associated with human cases of O157 (traceback premises). Sampling of such premises was thus biased toward finding O157 as compared to a more random sampling. For example, 7 (63.6%) of 11 dairies that were traced back and sampled for one study were found positive for O157 (Wells et al., 1991). Of 1,139

nontraceback dairies sampled in the same study and in two other studies, only 27 (2.4%) were positive (Wells et al., 1991; Hancock et al., 1994; Hancock et al., 1993b).

The greater the number of animals sampled on a premises, the greater the likelihood of that premises being found positive for O157. Studies which sampled at least 30 animals per premises found, on average, a higher prevalence of O157-positive herds than did studies which sampled fewer animals on each premises. For example, in a study of dairy cattle in Washington state in which almost 60 animals per premises were sampled, the herd prevalence was 8.3 percent (5/60) (Hancock et al., 1994). However, in the National Dairy Heifer Evaluation Project (NDHEP), a 1991 study of dairy calves in which an average of 6.5 animals per premises were sampled, the rate was 1.8 percent (19/1,068) (Hancock et al., 1993b).

Current research involving intensive repeated sampling in Washington state indicates that dairy herd prevalence may be in excess of 50 percent (Personal communication, D. Hancock, Washington State University, February 1994). There are premises, however, on which O157 has not been found despite repeated sampling.

What about O157 in cattle in other countries?

O157 has been found in bovines in six countries other than the U.S. (Table 3.2). Outside the U.S., the highest reported rates of isolation of O157 from samples of at least 100 cattle have been in Ontario, Canada, where 1.5 percent (3/200) of beef cattle were found positive at an abattoir and 1.7 percent (10/592) of dairy calves were positive on 80 premises (Table 3.3).

Throughout the world, the presence of O157 in cattle appears to be associated with the presence of the organism in humans. There is no report of any country finding O157 in its human population but not in its cattle population. In addition, no country is known to have found O157 in cattle but not in humans. Although no human cases have yet been reported from Egypt, O157 has been isolated from water buffalo there (Dorn and Angrick, 1991). Nine countries with human isolates of O157 have not reported testing cattle for the organism. Of the five countries other than the U.S. which have human isolates and have tested cattle (Argentina, Canada, Germany, Spain, and the United Kingdom), all have found O157 (Orskov et al., 1987; Clarke et al., 1988; Montenegro et al., 1990; Gonzalez and Blanco, 1989; Chapman et al., 1989). Thailand is the only country for which all published reports indicate negative findings for O157 in both human and cattle populations (Bettelheim et al., 1990; Suthienkul et al., 1990). Sri Lanka has not found O157 in its cattle population, but no reports could be found regarding human isolates (Mohammad et al., 1985; Mohammad et al., 1986). New Zealand had no published reports on O157, but has informally reported that O157 has not yet been found in either their human or cattle population (Personal communication, M. Muirhead, NZ Meat Products Board, July 1993). Most other countries apparently have tested neither their cattle nor their human population.

What is the pattern of O157 shedding in cattle?

Preliminary evidence suggests that cattle transiently or sporadically shed O157 in their feces and that the excretion period ranges from hours to weeks (Wells et al., 1991). Several long-term shedding studies are in progress to assess the endemicity of O157 in herds and to determine if and why the organism persists in some herds and not in others. In a completed study, dairy calves 4- to 8-weeks old were orally inoculated with O157 and periodically sampled to assess the duration and magnitude of O157 fecal shedding (Personal communication, W. Cray, NADC, Ames, IA, July 1993). O157 was still detected in the feces of some calves 5 weeks later. In another study, shedding was transient and clinical disease absent when 4 calves, aged 24 hours to 2 months,

were fed O157 (Personal communication, T. Besser, Washington State University, July 1993). The duration of fecal shedding ranged from 2 to 45 days, and plate counts were highly variable. Calves again shed O157 when reinoculated 1 week to 2 months following the last positive fecal sample.

What are the risk factors for O157 in cattle?

If O157 has established or expanded a niche in the intestinal flora of cattle during the last 15 years or so, it is reasonable to examine preharvest factors or practices which may have contributed to such a change. Many factors may affect the likelihood that cattle will carry O157 and thereby increase the potential that the organism will move into the slaughter and processing stages of the farm-to-consumer continuum.

Many of the potential risk factors may be related to stress. Stressors such as weaning, dietary changes, shipping, fasting, and changes in immunologic status and disease predispose animals to shifts in the indigenous microflora of the gastrointestinal tract. Numerous studies conducted on *Salmonella* have shown that outbreaks of *Salmonella* in domestic and other animals are often preceded or accompanied by stress factors (Brownell et al., 1969; Grau and Smith, 1974; Nottingham and Urselmann, 1961; Robinson, 1967; Robinson and Royal, 1971; Salisbury, 1958; Tannock et al., 1971).

Starvation of animals before oral inoculation with *Salmonella* lowers the number of organisms required to produce infection and increases the duration of *Salmonella* shedding in the feces. High concentrations of animals increase the risk of salmonellosis and shedding, as does holding animals in pens or yards, and overcrowding during transport and slaughter (Galton et al., 1954; Williams and Newell, 1968). Due to the similarities between *Salmonella* spp. and *E. coli* spp., it is reasonable to assume that shedding of O157 may also increase when animals are stressed.

Diet. Dietary factors are known to affect intestinal flora in both humans and animals (Copperstock and Zedd, 1983; Savage, 1982). This is true for ruminants as well as monogastrics. Studies have been conducted in an effort to determine the effects of stress on the indigenous microflora of the gastrointestinal tract in various species. Results of these studies are generally applicable across species. It is therefore plausible that when a calf is weaned, or other dietary stresses occur, the ability of O157 to successfully colonize may be enhanced.

The first notable dietary stress in an animal's life is weaning (Tannock, 1983). In one study, the number of *E. coli* organisms in the small intestine of pigs increased 3 to 7 days post weaning (Smith and Jones, 1963). In another study, weaned animals had higher numbers of *E. coli* organisms in the stomach, duodenum, and jejunum than did unweaned animals (Barrow et al., 1977). Stress associated with weaning may allow O157 to colonize the gastrointestinal tract of uninfected calves. If O157 is already present in the calf gastrointestinal tract, weaning could result in increased fecal shedding of the organism. Culturing animals for O157 during the weaning period may increase the probability of recovering organisms from the feces. Age-adjusted analysis of one study indicated that postweaned dairy calves were three times more likely to test positive for O157 than were preweaned calves ($p=0.005$) (Garber et al., 1994).

Starvation or abrupt changes in the diet of adult animals can also produce changes in the indigenous microflora of the gastrointestinal tract. *E. coli* numbers have been shown to increase in the gastrointestinal tracts of stressed and starved animals and birds (Byrne and Dankert, 1979; Morishita and Miyaki, 1979; Smith, 1965). Two bacterial populations are sensitive indicators of dietary stress: Lactobacilli decrease in number and coliform bacteria increase in number under conditions of dietary stress for the host (Albert et al., 1978; Cohen et al., 1967; Gracey and Stone,

1972; Gracey et al., 1973; Heyworth and Brown, 1975; Mata et al., 1972). The number of coliforms is thought to be regulated largely by the anaerobic members of the indigenous microflora of the large intestine. Volatile fatty acids produced in the large intestine by anaerobic bacteria inhibit the growth of coliforms (Lee and Gemmell, 1972; Schaedler et al., 1965). Dietary stress which alters the gastrointestinal ecosystem presumably influences the anaerobes present. Therefore, any decrease in the metabolic activities of the anaerobes will be reflected in a rise in coliforms.

Recent work has shown that O157 grows poorly in the ruminal environment of a well-fed animal (Rasmussen et al., 1993). However, the rumen contents of cattle held off feed for 48 hours did not inhibit the growth of O157. Thus, there is evidence that dietary stress may indeed influence the development, and presumably shedding, of O157 in cattle.

Tentative associations found between O157 prevalence and nutritional variables in two studies are inconclusive. In the NDHEP, early age of onset of feeding solids (forage and/or grain) to dairy calves resulted in slightly higher, but not statistically significant, prevalences of O157 shedding (Hancock et al., 1993b). The feeding of oats in the starter ration of dairy calves was also associated ($p=0.08$) with higher levels of O157 (Garber et al., 1994)

Computerized feeders. One study found that the use of computerized feeders was associated with the presence of O157 on dairy premises in Washington state (odds ratio $=8.8$, $p<0.05$) (Hancock et al., 1994). Such systems allow a prescribed composition and amount of feed to be dispensed to individual animals at preset intervals. Using such a system, dairy animals may be fed up to eight times per day rather than just twice. While it is likely that both the composition of rations and the timing of feedings have a potential effect on the intestinal flora of cattle, specific elements of the process which might increase the likelihood of O157 shedding have yet to be identified.

Subtherapeutic antimicrobials. Though there is temporal evidence that the use of pharmaceuticals in the cattle industry may be associated with the emergence of O157 as a human pathogen, there are few data to substantiate this hypothesized association. Use of pharmaceuticals in animals has increased since the early 1970's. Pharmaceuticals are used to promote healthier animals and more efficient production. Such use has raised concerns regarding antibiotic-resistant strains and selection of microorganisms not historically considered to be indigenous in these species.

Subtherapeutic antimicrobials fed to cattle to promote growth could conceivably alter the gastrointestinal tract microflora and allow O157 to establish itself. The spread of resistant organisms from farm animals to humans, rodents, flies, and other farm animals has been documented (Levy and Marshall, 1988). Other studies have shown that antibiotic-resistant organisms of farm origin can cause disease in humans (Hummel et al., 1986; Parsonnet and Kass, 1987; Fish et al., 1967; Lyons et al., 1980; Rowe et al., 1979; Spika et al., 1987; Ryan et al., 1987). There is no evidence to suggest that O157 has acquired resistance to antimicrobials. In fact, most O157 organisms are susceptible to a variety of antimicrobials.

In 1985, the National Cattlemen's Association recommended that the use of subtherapeutic antibiotics in cattle feed be stopped and antibiotics be used only to treat specific diseases. Since then, the use of antibiotics in cattle feed has been reduced. Only about 10 percent of all feed produced for beef cattle now contains antibiotics (Krause, 1992).

Ionophores. Carboxylic polyether ionophore antibiotics, more commonly known only as ionophores, are a class of compounds produced primarily by *Streptomyces* spp. (Bergen and Bates, 1984). Ionophores are used to improve feed efficiency in feedlot cattle and dairy heifers. Ionophore products are reported to be used in greater than 90 percent of feedlot and farmer feeder cattle,

increasing in use for stockers, and used in less than 50 percent of replacement heifers and beef and dairy calves (Personal communication, D. Tugen, Doane's Marketing Research, August 1993). Improved feed efficiency is due to a series of changes in the ruminant digestive process, including altered balance of ruminal volatile fatty acids, decreased methane production, and decreased degradation of rūminal protein (Anderson and Horne, 1987; Fischer et al., 1971; Rogers and Davis, 1982; Tyler et al., 1992).

Monensin, one of two major ionophore products used in cattle, was first approved for use in feedlot diets in 1975 and for use in cattle on pasture in 1978. Within 2 to 3 years after approval, monensin was used in essentially all large feedlot operations (>4,000 head). There has been little change in the usage of monensin in feedlot cattle since 1980 (Personal communication, D. White, Eli Lilly and Co., August 1993).

Because the implementation of feeding ionophores to cattle occurred just prior to the first human cases of O157 traceable to cattle in the early-1980's, there is concern that ionophores may alter the bovine gastrointestinal flora in a way that selects for O157. While ionophores do inhibit gram-positive organisms in the rumen, their effect in the lower intestinal tract is not clearly defined. *E. coli* and other gram-negative organisms such as *Salmonella* spp. are rarely found in the anterior portion of the intestinal tract, but reside in the lower portion of the tract. What effects ruminal changes might have on the lower gastrointestinal tract are not completely known.

There are no reports of the effects of feeding ionophores on the numbers and shedding of *E. coli* organisms. Based on studies done for Food and Drug Administration (FDA) approval, feeding of approved ionophores to cattle does not result in antimicrobial resistant strains of *E. coli* and does not impact on numbers or the duration of shedding of *Salmonella* spp. (Personal communication, D. Fagersburg, Colorado State University, August 1993). There are no data to support or refute the hypothesis that feeding ionophores could help to select for organisms not previously known to be indigenous to the bovine intestinal tract, such as O157. Although one study found that calves from farms that reported the use of ionophores in grain had a higher O157 prevalence than did calves from dairy farms that did not (5.5 vs. 2.3 per 1,000; $p=0.03$), a recent case-control study conducted on dairy farms found no association between the feeding of ionophores and O157-positive farms (Hancock et al., 1993b; Garber et al., 1994).

Herd size. Findings from the NDHEP indicated that dairy herd size does not significantly influence the prevalence of O157 in calves. Among a total of 1,062 dairy herds sampled, the prevalence of herds with O157 was similar among herds with less than 200, 200 to 499, and 500 or more cattle at 1.3, 1.5, and 3.2 percent, respectively (Hancock et al., 1993b). The slightly higher prevalence among large herds was expected, as the number of calves sampled increased with herd size. The overall prevalence of calves with O157 was also similar at 3.4, 3.2, and 4.5 positive calves per 1,000 sampled ($p=0.76$). Conversely, in a Washington state-based study in which 60 dairy herds were sampled, the five O157-positive herds were smaller in size than the negative herds (177 vs. 357 average cows per herd, $p=0.08$) (Hancock et al., 1994).

Calf contact with older cattle. The potential for cattle-to-cattle transmission of O157 was addressed in the NDHEP by comparing positive and negative herds with respect to contact with older cattle and time spent by calves in maternity pens. Prevalence of O157 among preweaned dairy calves having contact with older heifers was similar to that among calves having no contact (3.5 per 1,000 vs. 4.2 per 1,000, respectively) (Hancock et al., 1993b). Likewise, there was no significant difference in herd prevalence between herds that did and those that did not permit contact among calves and older animals ($p=0.41$). The length of time calves remained in the maternity area had no effect on O157 occurrence.

Housing. The types of housing used for dairy cattle and calves has changed somewhat in recent years. For example, the use of dairy calf hutches has increased by roughly 50 percent in the last 10 years, from about 24 percent of dairy premises in 1982 to 36 percent in 1992 (Hoard's Dairyman, 1993). However, the role that housing may play in O157 shedding among various types of dairy cattle is unclear.

A case-control study of O157 in Washington state found no significant association between premises with shedding dairy cattle and the use of pole barns (barns with no walls), the use of free stalls, or the practice of loose housing (Hancock et al., 1994). The NDHEP found no significant associations between the prevalence of O157 shedding by preweaned dairy calves and the use of group housing or the use of calf hutches (Hancock et al., 1993b). A follow-up case-control study has recently found that farms where dairy calves were housed indoors or as a group were more likely to test positive for O157 (Garber et al., 1994). In addition, on premises that housed dairy calves individually, grouping calves prior to weaning was more likely to result in O157 shedding than grouping calves after weaning.

Waste management. Changes in methods of waste management, especially in dairy herds, have taken place over the last 10 to 15 years. The use of waste lagoons and slurry irrigation of pastures and fields used to produce forage has increased greatly since the late 1970's. An annual survey conducted by *Hoard's Dairyman* magazine found that the percentage of dairy premises which own pumps used for liquid waste management has increased from about 9 percent in 1977 to roughly 28 percent in 1992 (Hoard's Dairyman, 1993). Previous survey results had revealed that the percentage of dairies that owned manure storage systems increased from less than 18 percent in 1981 to over 43 percent in 1991. The most common systems in 1991, and those with the largest increases in usage over 1981, were enclosure (solid or semisolid waste) and earthen basin (liquid waste) systems (Figure 3.1).

More information about the on-farm ecology of O157 is needed to more fully address the relationship between waste management practices and the presence or spread of O157. Epidemiologic studies have produced mixed results in finding associations between waste management practices and O157 shedding. A case-control follow-up study to the NDHEP found no association between the type of waste management on a premises and O157 shedding in calves (Garber et al., 1994). Conversely, a study in Washington state found an association between premises with dairy cows shedding O157 and the spreading of slurry on pastures (odds ratio = 7.0; $p<0.10$). The same study also found O157 to be associated with a shorter waiting period between application of slurry to pasture and grazing of livestock on pasture (12 vs. 44 days; $p=0.08$) (Hancock et al., 1994). Data collected through the NDHEP indicated an association between shedding in dairy calves and the use of wood product bedding (shavings or sawdust) (Hancock et al., 1993b). Such bedding is often used in conjunction with liquid waste handling systems.

Transportation and holding. According to a recent survey of packers, the amount of time spent in transport may have increased for certain types of cattle (cows and bulls), but decreased for others (fed steers and heifers) (USDA:APHIS, 1994). Seventy-five percent of respondents to the survey reported holding times of 12 hours or less. The maximum reported holding time was 24 hours.

As cited previously, the rumens of cattle held off feed have been shown to be less inhibitory to O157 than those of well-fed cattle. Starvation can lead to greater susceptibility to some enteric organisms and can prolong the period of shedding in the feces. Since cattle are routinely held off feed during transportation and prior to slaughter, this is a period during which an increase in the numbers of O157 in the gastrointestinal tract of cattle may occur. Such an increase could lead to spread of the organism from one animal to another and to contamination of the environment just prior to slaughter. It has been shown that the prevalence of certain enteric organisms can increase

when transportation and holding times are increased (Nottingham and Urselmann, 1961; Robinson, 1967).

Age. It has generally been found that the group of animals with the highest percentage of O157 shedders are postweaned heifers or calves (Table 3.1). In the two studies in which more than one age group of cattle was sampled for O157, the prevalence of the organism was higher in nontraceback dairy heifers or calves (up to 25.3 per 1,000) than in nontraceback adult cows (up to 3.7 per 1,000) (Wells et al., 1991; Hancock et al., 1994). The prevalence of O157 among dairy calves in the NDHEP increased from 2.9 per 1,000 at 2 weeks of age to 6.2 per 1,000 at 12 or more weeks of age, but the trend was not statistically significant (Hancock et al., 1993b). Postweaned heifers in five O157-positive dairy herds in Washington state had a higher prevalence (9.6%) than preweaned heifers (0%), dry cows (2.6%), and lactating cows (1.7%) (Hancock et al., 1994). In a study conducted on Wisconsin and Washington dairy farms, O157 was recovered significantly more often from fecal specimens in calves (2.3%) and heifers (3%) than adult cows (0.15%) (Wells et al., 1991).

Type of cattle. Prevalence studies offer no evidence that dairy cattle are more likely to shed O157 than are any other type of cattle. Among studies that have looked at a large number of samples ($\geq$1,000), the highest overall rate of O157 isolation was obtained from adult beef cattle in Washington state (0.7%) (Table 3.1). In the same study, feeder cattle had a prevalence (0.33%) slightly higher than that of adult dairy cattle (0.16%) (Hancock et al., 1994).

Mastitic cows. Mastitis, an inflammation of the udder, can be caused by a variety of bacteria including coliforms like *E. coli*. It has been postulated that mastitic cows are a possible source of O157 contamination of beef. Such contamination would presumably occur when mastitic cows are slaughtered. The available evidence, however, does not support the hypothesis that mastitic cows are associated with O157 in ground beef.

In a 1993 study conducted at the University of California, over 500 clinical cases of coliform mastitis from California, Arizona, Oregon, and Texas were evaluated for the verotoxin-producing gene of O157 (Cullor, 1993). No cases were associated with O157. A similar study at Pennsylvania State University also found no O157 in isolates of coliform mastitis (Personal communication, R. Wilson, *E. coli* Reference Center, Pennsylvania State University, September 1993).

Cows with clinical mastitis that go to slaughter are documented in data maintained by the FSIS. Most such cows are passed for human consumption following inspection. From 1983 to 1992, an average of 5.6 percent of all mastitic cows were condemned, most during postmortem inspection[3] (USDA:FSIS).

If mastitic cows are a major source of O157 contamination of ground beef, then, based on the premise that the prevalence of O157 has increased since the early 1980's, an increase in the prevalence of mastitic cows since that time might be anticipated. The annual rate of mastitic cows at slaughter has not increased over the past 10 years (Figure 3.2). Although the number of human O157 cases has been highest in the summer months (May through September), there appears to have been no seasonal difference in the rate of mastitic cows per 1,000 cows at slaughter (Figure 3.3) (Griffin and Tauxe, 1991). In addition, the number of mastitic cows at slaughter has decreased

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[3] Corrections were not available for two obvious errors in the data, one in the 2nd quarter of fiscal year 1988 and one in the 1st quarter of fiscal year 1990. In each case an average of the other monthly figures for that slaughter plant was substituted.

from 17,360 in fiscal year (FY) 1983 to 10,290 in FY 1992. The number of mastitic cows at slaughter by season has had a distribution similar to that of the rate of such cows at slaughter by season.

Nonambulatory cows. Some have hypothesized that nonambulatory cattle (downer cows) may be more likely to harbor and/or shed O157 than other cattle. It is estimated that more than half of all nonambulatory cows are rendered rather than slaughtered for human consumption and, in general, nonambulatory cattle make up a small percentage of the total number of cattle slaughtered (USDA:APHIS, 1993). Those that are slaughtered have an identifiable physical or noninfectious reason for being nonambulatory, such as a hip or leg injury. Because O157 is not known to cause clinical illness in cattle, it is unlikely to be a cause of cows being unable to stand. One hypothesis is that the possible increased use of antibiotics in nonambulatory cattle could help to select for O157 or allow O157 to more readily colonize such animals. While such a scenario is conceivable, most strains of O157 are susceptible to broad-spectrum antibiotics of the type likely to be given to nonambulatory animals. It is also possible that the stress resulting from the downer condition itself may increase the likelihood of shedding O157 if it is present. A current FSIS study of nonambulatory cattle should help determine if such animals are at greater risk of having O157 than are other cattle.

Season. Seasonal effects on O157 prevalence in cattle have not been fully elucidated. The NDHEP, which collected calf fecal samples from approximately equal numbers of dairy herds each month, showed no seasonal pattern in the identification of positive herds (Hancock et al., 1993b). Although cattle were sampled in other months, all five O157-positive dairy herds in a Washington state study were identified from June through September (Hancock et al., 1994). In addition, the isolation rate of O157 was highest during the summer months, reaching a peak of 13 positives per 1,000 samples in September.

Soiled hides or haircoats. Wet, muddy holding conditions prior to slaughter increase hide and haircoat contamination and may provide O157 with an additional mode of entry into the slaughter facility. Such animals could carry O157 on their hide or haircoat regardless of their fecal culture status. Contamination of a carcass might result directly from contact with the soiled hide or indirectly from contact with equipment, utensils, or workers contaminated by the hide. Some dehiding practices may aerosolize microorganisms and thus increase the potential for contamination of a carcass from a hide carrying O157.

Are there any factors associated with decreased shedding?

Epidemiologic studies of O157 have found that some factors are associated with a lack of shedding of O157. The feeding of whole cottonseed, both to adult cows and to heifers, has been associated with a lack of O157 shedding (Hancock et al., 1994; Garber et al., 1994). The feeding of clover as a first forage to calves and the grazing of calves on clover pasture were both associated with a lower likelihood of shedding (Garber et al., 1994). In addition, calves which received milk replacer had a lower prevalence of O157 shedding than those not receiving milk replacer (Hancock et al., 1993b).

Table 3.1. Sampling of Cattle for *E. coli* O157:H7 in the U.S.

Location (reference)	Period	No. and Type of Animals / Premises	Animal Prevalence (positives)	Herd prevalence (positives)	Age-group prevalence (per 1,000)	Comments
WI (Wells et al., 1991)	1986	226 dairy cattle 2 premises	2.2% (5)	100.0% (2)	Calves & heifers: 58.8 Cows: 0.0	Both premises were tracebacks[1]
		428 dairy cattle 11 premises	1.2% (5)	27.3% (3)	Calves & heifers: 25.3 Cows: 3.7	
		46 dairy cattle 1 stockyard	2.2% (1)	NA	Calves & heifers: 52.6 Cows: 0.0	Stockyard was a traceback[1]
WA/OR (Wells et al., 1991)	1987	539 dairy cattle 9 premises	1.3% (7)	55.5% (5)	Calves & heifers: 22.2 Cows: 0.0	All premises were tracebacks[1]
		27 dairy heifers & calves 1 packing house	0.0% (0)	NA	Calves & heifers: 0.0	Packing house was a traceback[1]
WA (Hancock et al.,1994)	1991 [a]	3,570 dairy cattle 60 premises	0.3% (10)	8.3% (5)	Calves: 0.0 Heifers: 6.5 Cows: 1.6	All positives found from June-Sept.
	1992 [a]	1,412 beef cows 25 premises	0.7% (10)	16.0% (4)	Cows: 7.1	
	1991-1992	600 feeder cattle 5 feedlots	0.3% (2)	40.0% (2)	Feeders: 3.3	
28 states (Hancock et al.,1993b)	6/91 to 5/92	6,894 dairy calves 1,068 premises	0.4% (19)	1.8% (19)	Calves: 3.6	No seasonal or regional patterns

[1] Premises believed to be potential sources of O157, based on traceback from human O157 cases

[a] Estimated period of sampling; actual period not reported

Table 3.2. Status of *E. coli* O157:H7 in Cattle in Other Countries

Country	Bovine Isolates?	Reference
Argentina	Yes	Orskov et al.,1987
Canada	Yes	Clarke et al.,1988
Egypt	Yes [1]	Dorn and Angrick,1991
Germany	Yes	Montenegro et al.,1990
New Zealand	No	*
Spain	Yes	Gonzalez and Blanco,1989
Sri Lanka	No	Mohammad et al.,1986
Thailand	No	Bettelheim et al.,1990
United Kingdom	Yes	Chapman et al.,1989

[1] Found in water buffalo; no report on cattle

* Personal communication, M.Muirhead, NZ Meat Products Board, 1993

Table 3.3. Sampling of Cattle for *E. coli* O157:H7 in Other Countries

Location (reference)	Period	Type of Sample	No. of Samples	No. Positive (%)	Comments
Argentina 4 premises (Orskov et al.,1987)	1977	Feces	13 calves	1 (7.7%)	Calves had diarrhea
Ontario, Canada abattoir (Clarke et al.,1988)	1987 [a]	Feces	200 dairy culls	1 (0.5%)	
			200 beef	3 (1.5%)	
			200 veal	0	
Ontario, Canada 80 premises (*)	1992 - 1993	Feces	1,478 dairy	14 head (0.95%) 8 prem. (10.0%)	Calves: 1.7% Cows: 0.45%
England abattoir (Chapman et al.,1989)	1987	Feces	207	2 [b] (1.0%)	
Germany (Montenegro et al.,1990)	1989 [a]	Feces	47 dairy	0	
			212 bulls	2 (0.9%)	
Galicia, Spain (Gonzalez and Blanco,1989)	1988 [a]	Feces	78 calves	1 (1.3%)	Calves had diarrhea
Sri Lanka (Mohammad et al.,1985)	1984 [a]	Feces	172	0	No sorbitol MacConkey agar
Thailand 2 premises (Suthienkul et al.,1990)	1988 [a]	Rectal swabs	66 beef	0	
			20 dairy	0	
Thailand abattoir (Suthienkul et al.,1990)	1988 [a]	Rectal swabs	10 beef	0	

* Unpublished data, S. Renwick, Agriculture Canada, 1993

[a] Estimated period of sampling; actual period not reported

[b] No H-types reported

Figure 3.1

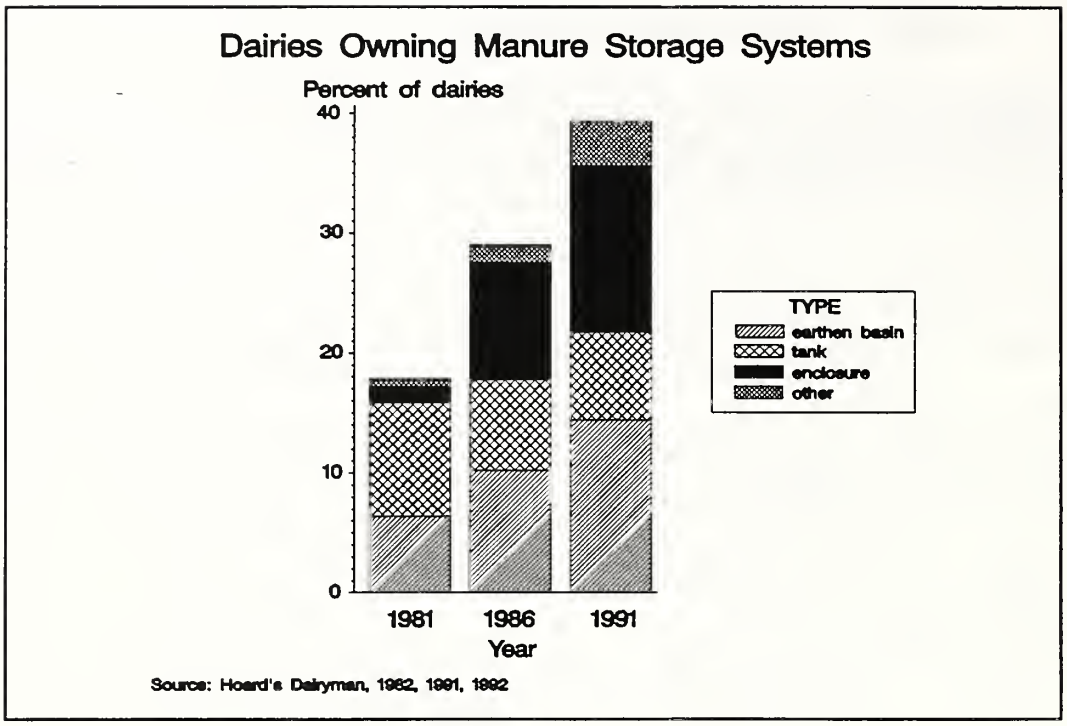


Figure 3.2

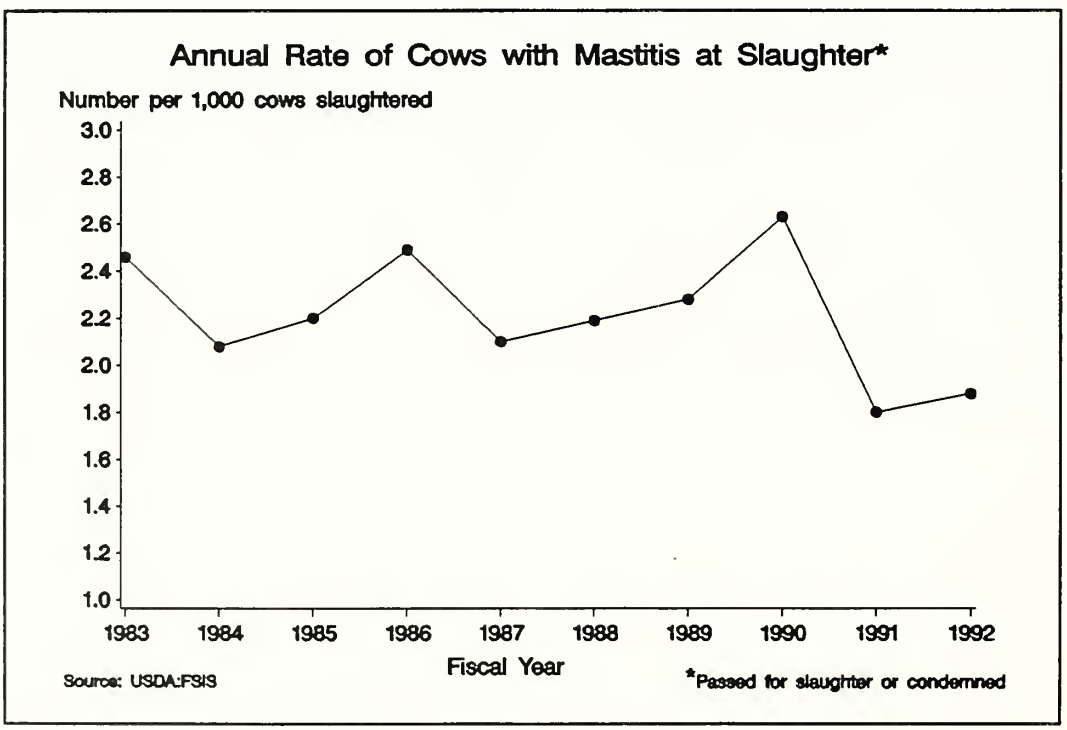
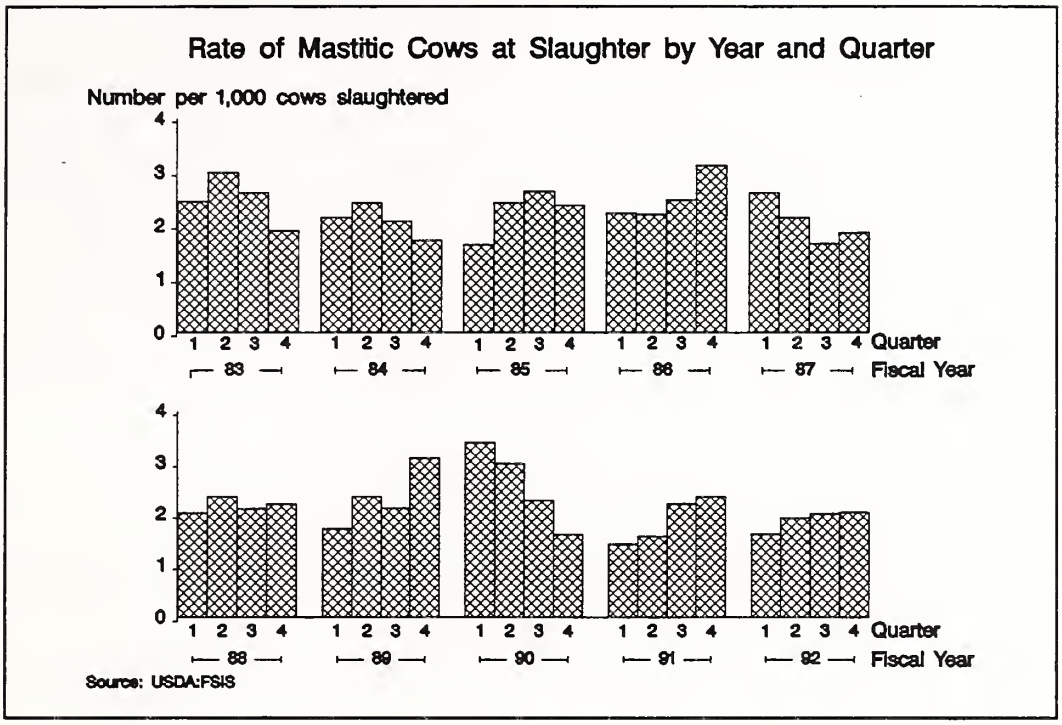


Figure 3.3


Rate of Mastitic Cows at Slaughter by Year and Quarter
Number per 1,000 cows slaughtered
4
3
2
1
0
1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 Quarter
83 84 85 86 87 Fiscal Year
4
3
2
1
0
1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 Quarter
88 89 90 91 92 Fiscal Year
Source: USDA:FSIS

E. coli O157:H7 in Human Food

What is the prevalence of O157 in food?

Very little direct sampling of foods of animal origin has been performed in the U.S. or in other countries (Tables 4.1 and 4.2). To date, there have been two large-scale samplings for O157 in the U.S. In one study, only 0.2 percent of over 2,000 steer and heifer carcasses were found positive for O157 (USDA:FSIS, 1994). Bob veal kidney was the item most frequently (0.5%) found with O157 in another study (Griffin and Tauxe, 1991). The organism has also been recovered from ground beef, poultry, pork, lamb, fancy veal kidney, and beef cuts. Outside the U.S., O157 has been isolated from ground beef in Canada and Germany, and pork in Canada (Table 4.2). It should be noted that none of the sampled products was associated with human disease and that some or all of the products could have been positive due solely to cross-contamination.

What human foods have been associated with O157?

Foods associated with human O157 infection include undercooked beef, raw milk, vegetables, apple cider, hot dogs, mayonnaise and salad bar items (Bryant et al., 1989; Griffin and Tauxe, 1991; Blake et al., 1983; Griffin et al., 1988; Ostroff et al., 1990; Belongia et al., 1991; CDC, 1993b; Pavia et al., 1990; Ryan et al., 1986; Padhye and Doyle, 1992; Martin et al., 1986; Riley, 1987; Herriott, 1993). Beef products included ground beef and roasts. Salad bar items included cantaloupe, pea salad and ranch dressing.

Raw Milk

What is the role of raw milk as a vehicle for O157?

Raw milk provides an excellent medium for disease-causing bacteria due to its nutrient content and near neutral pH. In the last 20 years, *Salmonella, Staphylococcus, Campylobacter, Yersinia*, and *E. coli* O157:H7 have been recognized as important agents of foodborne illness associated with raw milk (Table 4.3). During this time, over 8,400 cases of bacterial milkborne illness have been documented.

As compared to beef products, raw milk is a relatively less important vehicle for O157. Raw milk has been associated with 2 outbreaks that resulted in 14 confirmed human cases, whereas beef has been implicated in at least 15 human outbreaks and over 900 cases. Cases of O157 illness associated with raw milk in the U.S. and Canada have all occurred since 1986 (Herriott, 1993; Borczyk et al., 1987). The first U.S. outbreak associated with raw milk occurred in 1992. Reasons for the relatively recent emergence of O157 in raw milk-related outbreaks are unclear.

The mechanism by which raw milk becomes contaminated with O157 is unknown; however, fecal contamination associated with milking is presumed. Pathogen sources may include the farm environment, whereby microorganisms can contaminate the cows' outer udder, hide, or haircoat and then invade the udder to be shed in the milk. Other pathogen sources may include contaminated equipment used for milking, filtering, cooling, storing, and milk distribution, as well as infected farm workers.

How much raw milk is consumed and where is it sold?

Specific data on raw milk consumption in the U.S. are unavailable because they are included in statistics for total fluid milk. However, the availability of raw milk and its subsequent consumption is believed to be decreasing. An indicator of raw milk consumption is the total volume of milk and cream consumed on farms where it is produced. This value equaled 943 million pounds in 1980 and 507 million pounds in 1990 (USDA:NASS, a).

Most raw milk sales occur on the farm directly from the producer to the consumer. The volume of all milk (both raw and pasteurized) sold by producers directly to consumers has decreased over time. In 1991, producers in 20 states sold a total of 1 billion pounds of milk directly to consumers (USDA:NASS, a). The sales included those by producers who sold raw and pasteurized milk from their own herds and milk produced by institutional herds. In comparison, data for 1980 show that direct milk sales to consumers totaled 1.5 billion pounds.

In 1987, the FDA banned the distribution and sale of unpasteurized milk outside of the state in which it was produced. A recent FDA survey revealed that intrastate raw milk sales are permitted in 27 states and raw milk was sold in at least 18 states in 1992 (Figure 4.1). The 111 raw milk dairies in the U.S. constitute 0.06 percent of all dairies (Table 4.4). The 34.1 million pounds of raw milk reportedly sold to consumers in 1992 by the 111 dairies was 0.02 percent of the 148.5 billion pounds of milk produced in the U.S.

What is the population at risk from raw milk?

An estimated one to two percent of the U.S. population may be at highest risk of illness from O157 due to the consumption of raw milk[4]. Based on the human incidence of *Salmonella* linked to raw milk consumption, the Oregon Health Division estimated that 1.2 percent of that state's population may consume raw milk (*Oregonian*, April 28, 1993). Of those that consume raw milk, the elderly and the very young are at highest risk of severe illness. Based on a 1-day dietary recall, the elderly consumed 0.4 pounds of fluid milk per day (less than one cup) in 1988, or about half as much as young children (USDA:HNIS, 1993).

How can illness from O157 in raw milk be prevented?

Besides monitoring for fecal coliforms, raw milk can be treated to reduce the risk of O157. In one method, either hydrogen peroxide (H_2O_2) or H_2O_2-producing bacteria are added to raw milk to activate the naturally occurring lactoperoxidase system (LPS) (Farrag and Marth, 1992). The bactericidal properties of this system are believed to be due to oxidizing enzymes, with gram-negative bacteria being more susceptible than gram-positives. The LPS is inhibitory to O157 for 12 hours at 30°C (86°F).

Pasteurization of all milk sold for human consumption is the only known way to eliminate the risk of O157 infection from milk. When multiple strains of O157 were inoculated in raw milk at 100,000 cells per ml and subjected to a minimum holding time of 16.2 seconds, a 1 to 2 $\log_{10}$ reduction was observed at 60°C (140°F) and no survivors were found at temperatures above 63°C (145°F) (D'Aoust et al., 1988).

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[4] Estimate based on populations of young and elderly in rural areas of states that sold raw milk in 1992.

Efforts to prohibit the sale of raw milk or to mandate pasteurization have been unsuccessful, as public demand is high in some states and raw milk consumers believe they are consuming a more natural, wholesome product. Washington state requires that a conspicuous sign be posted near retail raw milk that states: "Warning: raw milk or foods prepared from raw milk may be contaminated with dangerous bacteria capable of causing severe intestinal illness. Contact your local health department for advice or to report a suspected illness." To date, Washington has not documented any O157 cases linked to raw milk.

O157 patients in Oregon have mentioned that if they had known that raw milk could cause disease, they would not have consumed the product. The state of Oregon recently passed a law that requires a label for raw milk sold at the retail level: "This product has not been pasteurized... may contain disease-producing organisms" (Personal communication, D. Herriott, USDA:APHIS:VS, Salem, OR, March 1994). Mandatory raw milk labeling in Oregon and other states may serve to protect consumers and reduce O157 incidence by providing them with important health risk information.

Currently, the FDA is evaluating the benefits and risks of raw milk sales (Personal communication, M. Headrick, FDA, Washington, DC, August 1993). In addition, the National Association of Federal Veterinarians recently approved a resolution which calls for the American Veterinary Medical Association to encourage states to require pasteurization of all milk sold (JAVMA, 1993). If the incidence of raw milk-associated O157 cases continues to increase, a ban on sales, mandatory pasteurization, or more frequent O157 testing may be in the best interest of public health.

Ground Beef

What is the role of ground beef in the transmission of O157?

In the U.S., ground beef products have been associated with at least 12 outbreaks of O157, far more than any other vehicle. Exact mechanisms for the contamination of ground beef are still unclear, but potential pathogen sources include: infected humans; fecal contamination of carcasses at slaughter; mixing with other contaminated product; and contaminated equipment used for mixing, grinding, storage, or final preparation. Although 0157 is a surface contaminant on beef carcasses and subsequently on the surface of cuts of beef, the grinding process mixes surface material throughout a ground beef product. Cooking the surface of beef cuts is likely to kill 0157, but ground beef must be cooked to a higher internal temperature because of the possibility of O157 contamination in the interior.

What are the consumption levels and trends for ground beef?

For the purposes of this paper, consumption is defined as the average quantity of a food eaten in a day and usage is the proportion of a specified group of people that consume a food during a limited time period. Disappearance[5] is used as an indicator of consumption (i.e., higher disappearance suggests higher consumption). Per capita disappearance of all beef peaked in 1976 and has since declined (Figure 4.2). Much of the decline has been in beef cuts and processed beef, which have fallen by 60 and 45 percent, respectively. In contrast, per capita ground beef disappearance

[5] Disappearance = (total production + beginning stocks + imports) - (exports + shipments to U.S. territories + ending stocks)

increased 21 percent from 24.7 pounds in 1980 to 29.8 pounds in 1991, after decreasing between 1976 and 1979 (AMI, 1992).

In 1977-78 and again in 1987-88, the Nationwide Food Consumption Survey (NFCS) tracked consumption and usage of two forms of ground beef: 1) ground meat such as meatballs or casseroles; and 2) hamburgers (Pao et al., 1982; USDA:HNIS, 1993). According to the surveys, the consumption of ground beef, excluding hamburgers, appeared to decline in all age groups over the 10-year period (Figure 4.3). The largest absolute declines tended to be in the 15-to-64 age range. Usage of ground beef, excluding hamburgers, decreased in all sex and age groups. Decreases in usage were fairly evenly distributed among the sex and age groups (Table 4.5).

In contrast, consumption of hamburgers, which included the weight of patties, bun, and condiments, increased in most age groups from 1977 to 1987 (Figure 4.4). Usage of hamburgers increased in all age groups (Table 4.5). The increase in both consumption and usage of ground beef in the form of hamburgers suggests that consumption patterns are shifting away from ground beef in other forms.

There are differences in consumption of beef by season and by region. NFCS data from 1977-78 indicate that per capita weekly home beef consumption was higher in the summer quarter than in other quarters of the year, purportedly reflecting more outdoor cooking of ground beef and steaks (McCoy and Sarhan, 1988). The percentage of beef consumed as ground beef was similar in all seasons (about 37%). Of four regions, the North Central had the highest beef consumption and the South had the lowest. In addition, the North Central region had a slightly higher percentage of beef consumed as ground beef than did the other three regions (39% vs. 32 to 37%) (McCoy and Sarhan, 1988).

Are the young and the elderly at greater risk of illness because they consume more ground beef?

Because of the higher rate and greater severity of O157 illness among the very young and the elderly, the usage levels and the usage and consumption trends of these risk groups are of interest. The data indicate that the young and the elderly are not at greater risk of O157 than are other age groups because of higher usage levels or increasing trends in consumption or usage of hamburgers. Their increases in these categories, and their overall usage levels, have generally been less than those of the middle age groups (Table 4.5; Figure 4.4). In addition, usage levels and decreases in consumption and usage of ground beef (excluding hamburgers) for the young and the elderly have generally been similar to those of other groups (Table 4.5; Figure 4.3).

Is ground beef consumption increasing outside of the home?

Consuming food outside of the home affords the consumer less control over food handling practices and, ultimately, safety of the food. However, hotels, restaurants, and institutions (HRI) are commanding an increasingly large proportion of total food expenditures as Americans eat outside the home more often. Food expenditures outside the home accounted for 34 percent of disposable[6] income spent on food (5.5% of total disposable income) in 1970 and increased to over 45 percent (6.1% of total disposable income) by 1990 (Figure 4.5). The market share of fast food increased from 20.1 to 27.3 percent of restaurant sales from 1980 to 1984, and grew more slowly for the rest of the decade and into the early 1990's to 31.4 percent (Figure 4.6). Hamburgers now

[6] Disposable income is personal income minus personal tax and non-tax payments.

account for about 47 percent of fast food sales or about 15 percent of all HRI sales (Bureau of Foodservice Research, 1992).

In a recent survey, 98 percent of households responded that they had eaten away from home in the previous month (Bureau of Foodservice Research, 1992). Hamburger fast food restaurants were the most likely place to have been patronized (78.5%) followed by pizza establishments (53.7%). Over one-fifth of the respondents reported eating more often in hamburger fast food restaurants in 1992 versus 1990. Hamburgers/cheeseburgers were the most commonly ordered item followed by french fries and steaks. Single parent families and all families with a child under age 13 ordered burgers, french fries, and pizza more often than other demographic groups. On average, adult males ate out more frequently (4.7 times/week) than did children/teens (4.2 times/week) or adult females (3.8 times/week).

How can consumers prevent illness from O157 in ground beef?

Although many outbreaks of O157 have involved ground beef prepared outside the home, it is likely that a large number of the sporadic and often unreported cases of O157 result from consumption of ground beef prepared at home. Thus it is important for everyone to pay strict attention to proper storage and handling procedures in order to limit the risk of acquiring O157 infection through consumption of ground beef. The raw meat should be refrigerated until immediately prior to cooking and should be kept separate from other foods during preparation. Knives and cutting boards used in preparation of raw ground beef should be washed thoroughly before use on other foods.

Even with these precautions taken, thorough cooking remains the final necessary step for elimination of O157 from contaminated ground beef. The cooked meat must be gray or brown throughout without any pink remaining. Juices from the ground beef should run clear. Several states have implemented regulations requiring restaurants to cook ground beef to an internal temperature of 68°C (155°F). Cooked meat should not be allowed to stand at room temperature and should be refrigerated if not eaten promptly.

Table 4.1. Sampling Foods of Animal Origin for *E. coli* O157:H7 in the U.S.

Location (reference)	Sampling Period	Type of Food	Number of Samples	Number Positive (%)
Madison, WI groceries (Doyle and Schoeni, 1987)	6/85 to 7/86	Ground beef	147	1 (0.7)
		Poultry	257	4 (1.6)
		Pork	250	3 (1.2)
		Lamb	200	4 (2.0)
U.S. (Griffin and Tauxe, 1991)	1987 to 1989	Bob veal kidney	1,478	7 (0.5)
		Fancy veal kidney	3,475	2 (0.06)
		Beef	1,668	2 (0.12)
		Chicken	3,977	0
U.S. ~100 slaughter plants (USDA:FSIS, 1994)	10/92 to 9/93	Steer and heifer carcasses	2,081	4 (0.19)

Table 4.2. Sampling Foods of Animal Origin for *E. coli* O157:H7 in Other Countries

Location (reference)	Sampling Period	Type of Food	Number of Samples	Number Positive (%)
Alberta, Canada Calgary supermarkets (Doyle and Schoeni, 1987)	6/85	Ground beef	17	5 (29)
		Pork	14	1 (7.1)
		Poultry	6	0
		Lamb	5	0
Manitoba, Canada Winnipeg retail outlets & restaurants (Sekla et al., 1990)	1988	Ground beef	71	0
	1989	Ground beef	165	4 (2.4)
Germany (Bulte, 1991)	1987 - 1991 [a]	Various	424	1 ground beef (0.24)
Thailand 10 markets (Suthienkul et al., 1990)	1988	Pork	111	0
		Beef	93	0
		Chicken	107	0

[a] Estimated period of sampling; actual period not reported

Table 4.3. Selected Bacterial Outbreaks Associated with Raw Milk, 1974-1993

Country and Year(s)	Agent	Cases[1]	Deaths	Reference
United States (1974)	*S. dublin*	74	16	D'Aoust,1989
Canada (1975)	*Y. enterocolitica*	58	0	deGrace et al.,1976
Australia (1976)	*S. typhimurium*	>500	0	D'Aoust,1989
Scotland (1976)	*S. dublin*	>190	0	D'Aoust,1989
United States (1976)	*Campylobacter* sp.	4	0	Bryan,1983
United States (1977)	*Salmonella* sp.	8	0	Bryan,1983
United States (1978)	*Campylobacter* sp.	3	0	CDC,1978
Scotland (1979)	*Campylobacter* sp.	648	0	Wallace,1980
England & Wales (1978-80) [a]	*C. jejuni*	>4,500	0	Robinson and Jones,1981
Scotland (1980-88)	*Salmonella* sp.	1,268	2	Sharpe,1989
Switzerland (1981)	*C. jejuni*	>500	0	D'Aoust,1989
United States (1980-81) [b]	*Campylobacter* sp.	>390	0	CDC,1981a; CDC,1981b; Potter et al.,1983
United States (1980-81)	*Salmonella* sp.	200	0	Bryan,1983
England & Wales (1984)	*S. zooepidemicus*	12	8	D'Aoust,1989
Canada (1984)	*C. jejuni*	9	0	*Disease Surveillance*,1984
United States (1984)	*C. jejuni*	12	0	CDC,1984
Canada (1986)	*E. coli* O157:H7	48	0	Borczyk et al.,1987
United States (1992-93) [c]	*E. coli* O157:H7	14	0	Herriott,1993
TOTALS		>8,400	26	

[1] Includes confirmed and presumptive cases
[a] 13 outbreaks
[b] Three outbreaks in four states
[c] Two outbreaks in one state

Table 4.4. Comparison of Dairy Data: Raw Milk States* and the U.S.

	Raw Milk States*	U.S.	%	Reference
Dairy Farms	1982: 80,643 1992: 54,300	1982: 277,762 1992: 171,560	29.9 31.7	USDC:BC,1989; USDA:NASS,1993a
Milk Cow Population (millions)	1982: 4.0 1992: 4.1	1982:10.8 1992: 9.8	37.0 41.8	USDC:BC,1989; USDA:NASS,1993b
Raw Milk Dairies, 1992	111	N/A	0.06	FDA,1993
Total Milk Produced (billion lbs)	1983: 55.7 1992: 66.8	1983: 139.6 1992: 148.5	39.9 45.0	FDA,1993; USDA:NASS,1990
Raw Milk Sold, 1992 (million lbs)	34.1	N/A	0.02	FDA,1993

* Producers from 18 states reporting raw milk sales in 1992
N/A = not applicable

Table 4.5. Percent of Respondents Using Ground Beef or Hamburgers, 1977 vs. 1987

Age Group and Sex*	Ground Beef			Hamburgers		
	1977	1987	Change	1977	1987	Change
Under 1 (B)	3.3	2.0	-1.3	0.0	1.0	+1.0
1-2 (B)	35.8	25.5	-10.3	7.1	12.6	+5.5
3-5 (B)	39.2	27.5	-11.7	9.4	13.9	+4.5
6-8 (B)	41.5	40.0	-1.5	10.8	16.4	+5.6
9-14 (M)	45.2	35.0	-9.8	13.1	35.3	+22.2
9-14 (F)	41.4	35.9	-5.5	12.9	25.2	+12.3
15-18 (M)	43.5	40.1	-3.5	19.0	39.4	+20.4
15-18 (F)	38.3	29.3	-9.0	15.1	30.4	+15.3
19-34 (M)	40.1	27.8	-12.3	18.0	27.5	+19.5
19-34 (F)	34.4	23.7	-10.7	11.6	18.0	+6.4
35-64 (M)	37.1	30.5	-6.6	10.4	18.2	+7.8
35-64 (F)	32.9	24.3	-8.6	5.2	12.3	+7.1
65-74 (M)	31.3	27.9	-3.4	3.6	9.2	+5.6
65-74 (F)	29.5	24.7	-4.8	3.2	4.1	+0.9
75+ (M)	32.8	25.8	-7.0	2.1	4.0	+1.9
75+ (F)	30.4	26.8	-3.6	1.2	4.3	+3.1
All Ages (B)	36.3	NA*	---	10.0	NA*	---

* B=both sexes, M=males, F=females, NA=data not available
Source: Pao et al.,1982; USDA:HNIS,1993

Figure 4.1

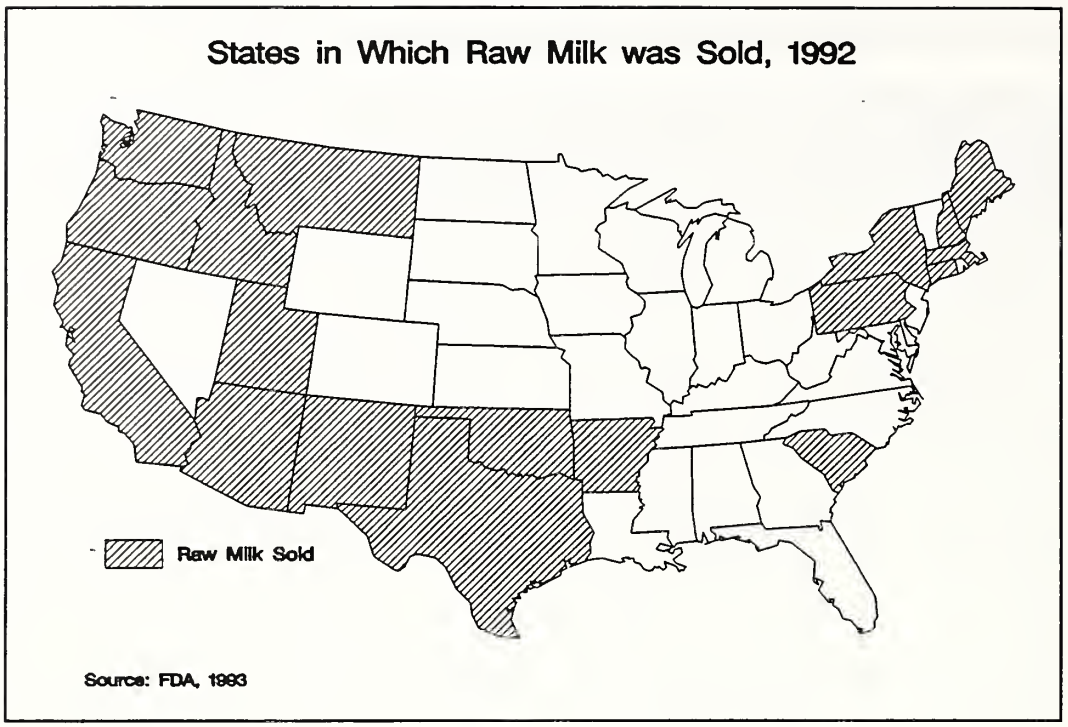


Figure 4.2

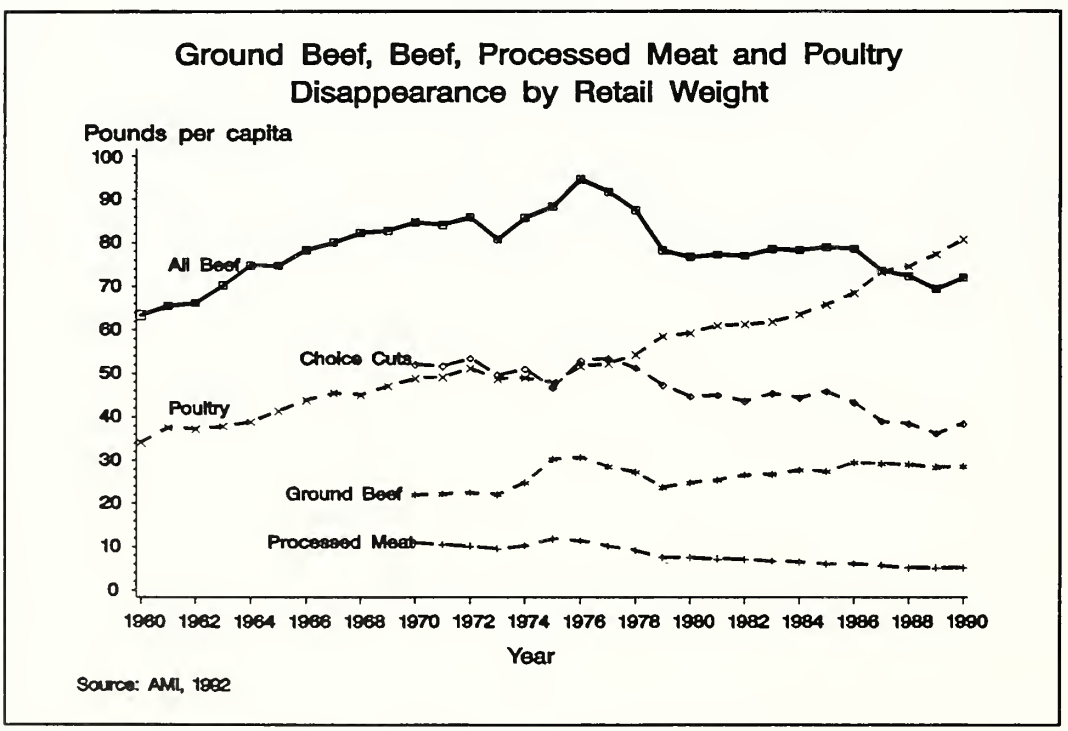
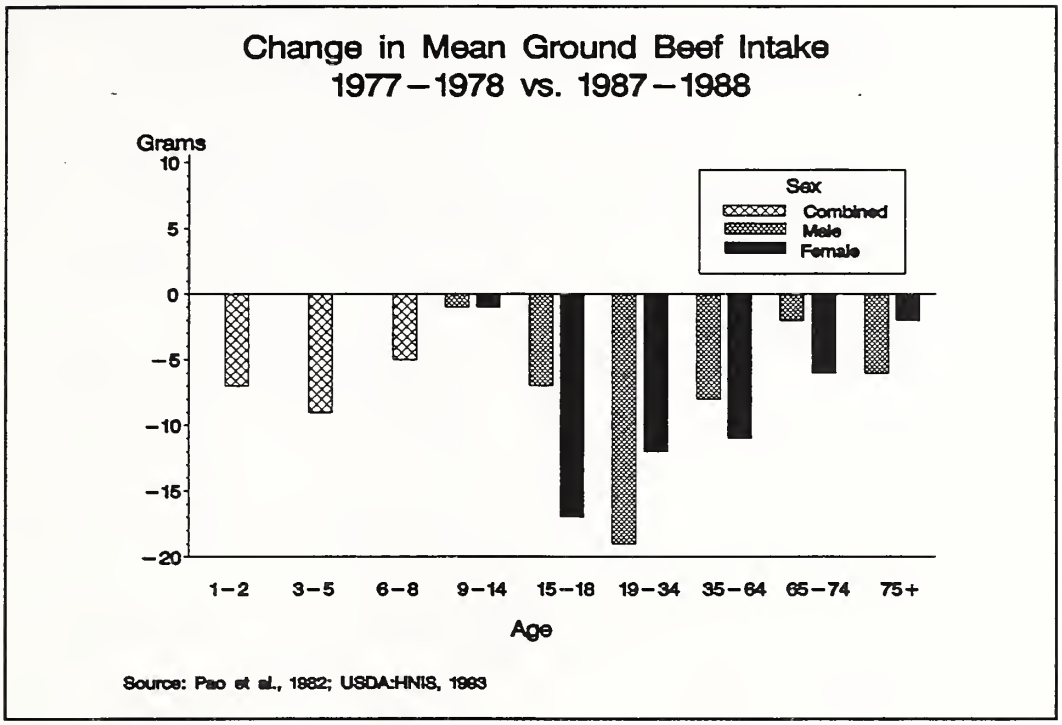


Figure 4.3



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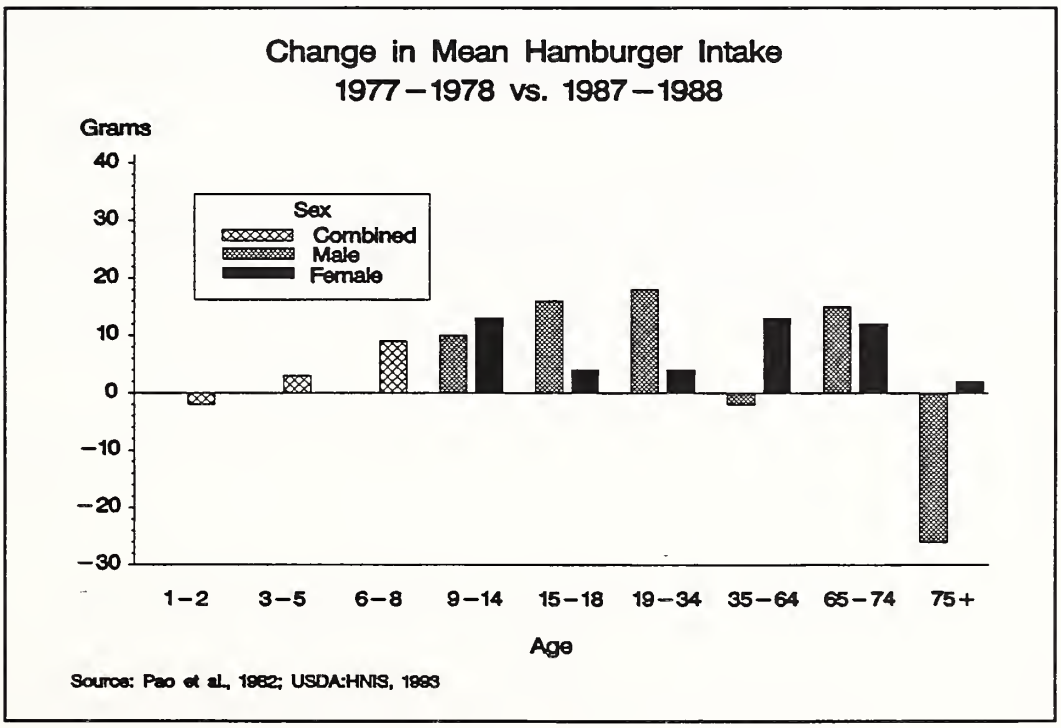


Figure 4.5

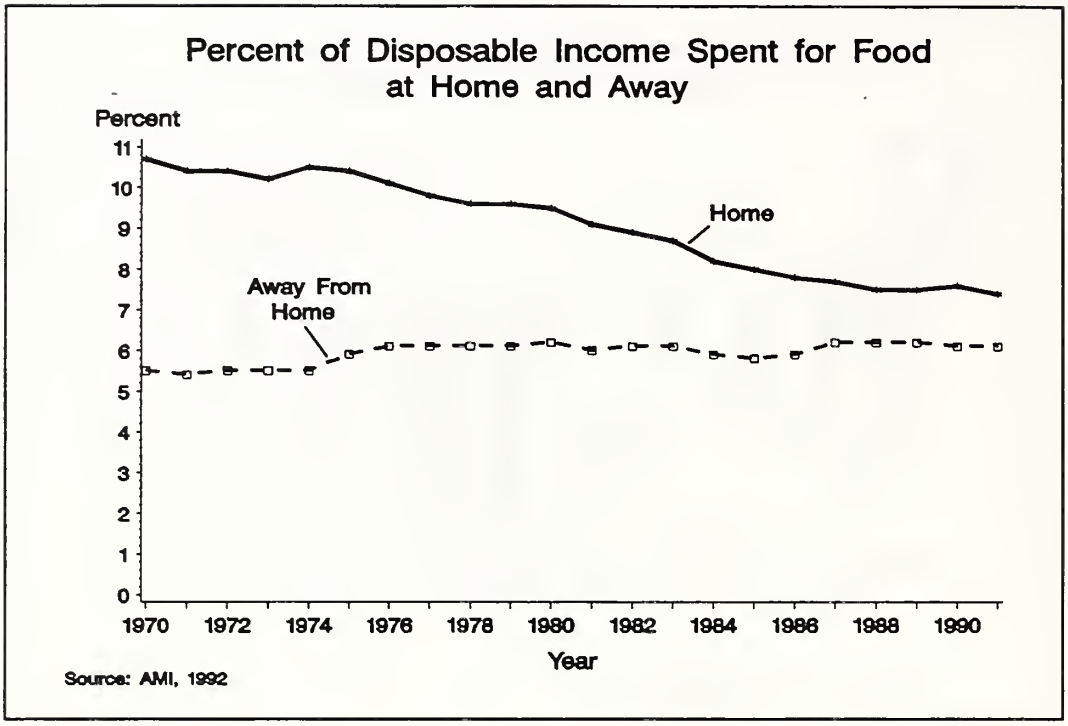
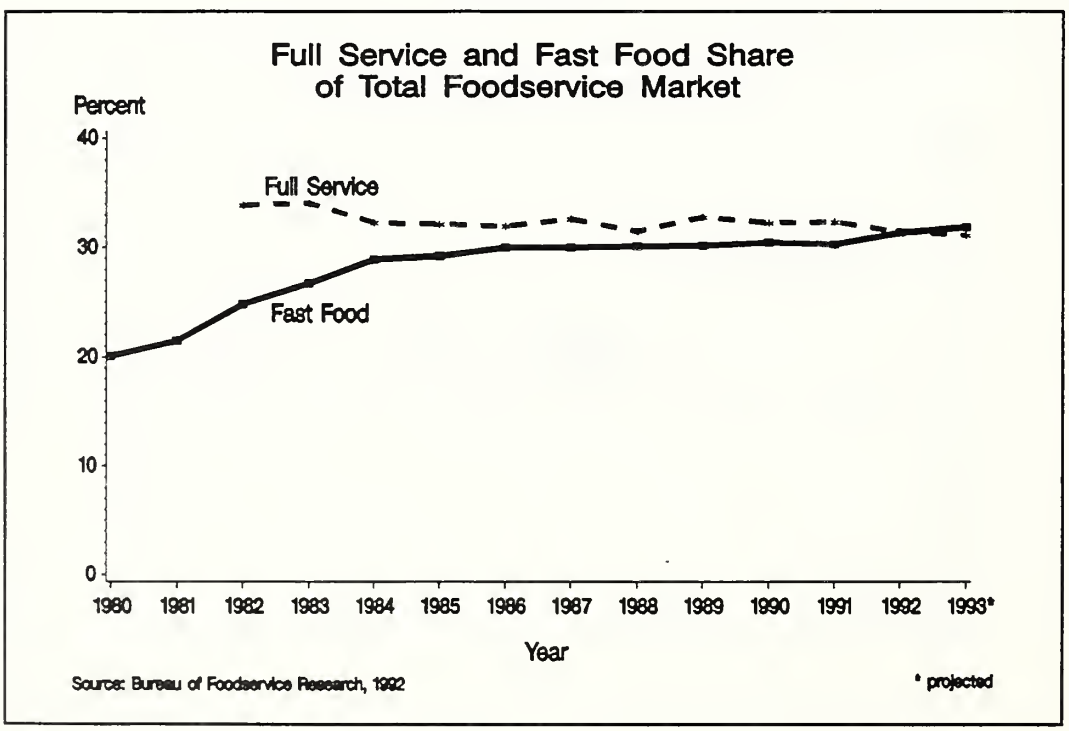


Figure 4.6



Ground Beef Production

Why do we need to know about ground beef production?

Ground beef products have been implicated in many human cases of O157, but the source(s) of contamination of these products has been difficult to trace. In order to better assess potential contributors to ground beef contamination, it is necessary to examine preharvest marketing channels and slaughter patterns for domestic cattle, availability of beef for ground beef production and postharvest marketing channels for ground beef.

Beef production in the United States consists of two main components: cow slaughter, and steer and heifer slaughter (Crom, 1988). Steers and heifers account for the largest share of the U.S. market, 82 percent. Beef and dairy cows supply 13 percent of the market. In the following discussion, the components of beef production are considered in terms of changes over time in the location and scale of production, and the movement of cattle and calves between the various stages of production. Calf slaughter is not discussed because calves usually move into processing channels that are separate from other cattle and because price differentials between veal and beef do not make it cost-effective to use calves in ground beef.

Are cow-calf operations becoming more concentrated?

After reaching 39.2 million head in 1982, the U.S. inventory of beef cows declined by 15 percent over the remainder of the 1980's (Figure 5.1). While all regions experienced declines in total numbers of beef cows, the largest percentage reductions of 23 and 11 percent, respectively, occurred in the Northeast and the North Central states, where only 16 percent of all U.S. beef cows are held. The Great Plains, which holds 38 percent of the U.S. herd, reduced its inventory by 9 percent between 1980 and 1991. Southern and western states, in which 45 percent of all U.S. beef cows are located, saw smaller reductions in their numbers, with declines of 7 and 5 percent, respectively. Thus, the trend in the 1980's was a further regional concentration of U.S. beef cows (Krause, 1992).

During the same period the shift toward larger average herd sizes also continued. From 1978 to 1987, the percentage of U.S. beef cows held in herds with 100 or more head increased by almost 7 percent. Marked declines in the number of herds with less than 20 head took place in all regions, with the U.S. percentage falling by 23 percent. The number of herds of between 20 and 99 head fell by 41 percent, with the North Central and Great Plains regions showing the greatest percentage decreases in number of herds of this size class (Krause, 1992).

When compared with the rate of structural change in other farm production subsectors, the changes in the beef cow-calf industry are minor and slow. In particular, the modest changes in beef raising enterprises contrast with the more dramatic locational and structural shifts in dairy production and cattle feeding.

Are similar trends occurring in dairy operations?

The number of milk cows in the U.S. decreased by 6 percent from 10.8 million head in 1980 to 10.2 million head in 1991 (Figure 5.1). In the North Central states, where 40 percent of milk cows are located, numbers declined by 11 percent. Steeper percentage declines of 18 and 13 percent were recorded for the South and the Northeast, respectively. These states have 31 percent of the U.S. milk cow inventory. In contrast, milk cow numbers increased by 27 percent in the western

states as population shifts and milder climates fueled greater specialization in production (Fallert et al., 1990).

Between 1978 and 1987 there were also dramatic shifts toward larger dairy herd sizes. The number of dairy herds with more than 100 head rose from 5 to 10 percent of all farms with milk cows. The number of milk cows on these larger farms increased from 31 percent of the total U.S. herd to 42 percent. Great Lakes states showed the strongest trend in moving from herds of less than 100 milk cows to herds of more than 100 milk cows (Fallert et al., 1990).

How do cull cows move to slaughter?

Data recording the movement of cows through markets to slaughter combines both beef and dairy cows as well as bulls, so it is not possible to distinguish different paths for the two types of cows. However, it is known that dairy cows and beef cows are slaughtered in approximately equal numbers; they accounted for 9.0 and 8.8 percent, respectively, of all cattle slaughtered in 1992 (USDA:NASS, b).

Total cow slaughter increased by 39 percent from 1980 through 1984 and then fell back to the earlier level by 1991. Culling rates for both beef and dairy cows increased between 1980 and 1991. For beef cows, the number of replacement heifers increased as a percentage of the beef cow inventory such that the average number of years in the herd fell 6 percent from 6.3 years to 5.9 years. The time dairy cows spent in the herd declined 8 percent, from an average of 2.6 years to an average of 2.4 years (USDA:NASS, a).

Approximately 40 percent of culled cows and bulls nationally were marketed through nonpublic markets directly to slaughter establishments in 1990 (Figure 5.2). The remaining majority moved through public markets such as auctions and terminal markets[7] on their way to slaughter. While the percentage of cows and bulls marketed directly increased during the 1980's, data on the 5 largest regional markets, which account for 83 percent of cow and bull marketings, indicate a mixture of trends (USDA:P&SA). It appears that the higher volume regions are marketing more cows and bulls directly, while the lower volume regions are marketing increasing percentages of their cows and bulls through auctions and terminal markets.

Changing marketing patterns may be attributable to the concentration of the cow slaughter industry into fewer, more centralized plants. In FY 1992, 90 percent of all cows were slaughtered in 68 plants, down from 152 plants in 1983. Because most cow slaughter plants are located in the heavier production areas, producers in these regions may have increased opportunities to sell directly to packers. Producers in lower production regions, however, must depend on auctions where culled cows are gathered and shipped greater distances to slaughter. The majority of packers in a recent survey reported increased transportation distances for cows in the last 10 years (USDA:APHIS, 1994). Over 70 percent of the packers that slaughtered cows or bulls reported receiving animals from distances of greater than 150 miles.

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[7] McCoy and Sarhan (1988) discuss the differences between livestock auctions and terminal markets. There is a large number of auctions throughout the U.S., although that number is decreasing. They usually operate once or twice a week and have one commissioned agent. Prices are determined by open public bidding. In contrast, there are only a few terminal markets in the U.S., most of which are concentrated in the Midwest. Terminal markets usually operate 6 days per week with a number of commissioned agents. Prices are set by private treaty.

How do calves and cattle move to backgrounding or feedlots?

The marketing channels for calves destined for beef production are more complex than those for cull cows (Figure 5.3). Once beef calves are weaned, they enter a grow-out phase (also called backgrounding) which lasts approximately 5 months. The availability and quality of forage, price outlook, and feedlot schedules influence the duration of backgrounding and the amount of weight gain. Whether the cow-calf operators continue to hold the weaned calves during this grow-out stage of production depends on the availability of forage on the farm or ranch (Krause, 1992).

Calves may first move through auctions and terminal markets on their way to backgrounding and then again when they are joined with other yearlings for further sale to feedlot operators (Figure 5.3). One source estimated that 70 to 80 percent of calves are moved through auctions to be fed out or grazed by a backgrounder (Personal communication, M. O'Conner, USDA:AMS, 1993). Of those backgrounded, 60 percent would again pass through auctions on their way to feedlots. However, the importance of these public markets, while varying regionally, is thought to be decreasing (Personal communication, S. Reed, USDA:ERS, 1993).

Regional movements of calves and cattle during these stages of beef production are difficult to track. Data are incomplete and patterns of movement vary from year to year depending on the availability of forage. The trends discussed here are derived from data recording state outshipments without destinations listed and from data recording state inshipments without points of origination stated. As a result, the complexity of regional movement known to exist, particularly for backgrounded animals, is not adequately conveyed.

In 1980, calf shipments out of state were concentrated in the South and the northern Great Plains states. Cattle outshipments occurred from states within and surrounding the Great Plains. Cattle inshipments for feeding and breeding focused on the southern Plains states but also included four western and two North Central states. The pattern of outshipments for 1990 is very similar to 1980, but state inshipments are more concentrated in the Great Plains (USDA:NASS, a). In addition, the number of feeder cattle imported from Canada and Mexico increased from 667,000 in 1980 to 3 million in 1990 (USDA:ERS, a).

What about concentration in the fed cattle industry?

Steers and heifers are typically placed in feedlots at weights between 700 and 850 pounds. They are held in closely confined pens for an average of 6 months and fed high energy rations to increase muscle and marbling qualities (Krause, 1992). The January 1 inventories of cattle on feed for 1980 and 1991 varied by only 3 percent, but their regional distributions were markedly different. Reductions of 13 to 35 percent occurred in the North Central, the West, and the South. Increases of 19 and 25 percent took place in the Northeast and the Great Plains, respectively (USDA:NASS, a). These patterns coincide with the changes in state inshipments discussed in the previous section.

With the geographical concentration of cattle on feed has come a concentration of cattle in larger feedlots. Although the number of feedlots with greater than 1,000 head capacity decreased by 10 percent from 1980 to 1991, the number of cattle marketed by these feedlots increased by 19 percent during the same period. In addition, feedlots with more than 32,000 head capacity in Colorado, Kansas, Nebraska, and Texas increased in number by 52 percent while the number of cattle marketed by these feedlots rose by 73 percent (USDA:NASS, c).

In recent years, attention has focused on the percentage of fed cattle marketed that are so-called captive supplies of major packing firms. These captive supplies are either owned and fed by the packing firms themselves or are purchased from feedlot operators under forward contracting or marketing agreements. The purpose of these arrangements is to assure slaughter plants a steadier supply of animals. The percentage of cattle slaughter fed by packers was 3 to 4 percent throughout the 1980's and the contracted percentage of slaughter ranged from 14.3 to 22.4 percent of the top 15 firms' supplies in the late 1980's and early 1990's.

How do fed cattle move to slaughter?

A larger percentage of steers and heifers is marketed directly to packers than of any other cattle type. The U.S. average rose from 88 to 94 percent between 1980 and 1990. This increasing trend held in 4 of the 5 regions in which more than 96 percent of the steers and heifers were marketed nationally (Figure 5.4). This closer marketing relationship is believed to have resulted from the increased size of feedlots and slaughter facilities plus the closer proximity of slaughter facilities to feedlots.

Consolidation of steer and heifer slaughter is especially pronounced. In 1980, the 4 largest firms slaughtered 35.7 percent of all steers and heifers, compared with 71.6 percent in 1990. Facilities that slaughter primarily fed steers and heifers are usually located close to feedlots. Thus, fed cattle, in general, do not travel as far immediately prior to slaughter as do cows or bulls (Personal communication, R. Mucklow, Western States Meat Association, August 1993). Results of a recent packer survey revealed that less than 50 percent of packers that slaughtered steers or heifers received them from a distance of more than 150 miles (USDA:APHIS, 1994). Transportation distances appeared to have changed little in the last 10 years.

What is the composition and availability of domestic beef for ground beef production?

The fat content of ground beef is an important determinant of composition. Different types of cattle contribute specific lean content manufacturing-grade meat and trimmings to the ground beef formulation. In general, cows and bulls contribute leaner meat and fed beef contributes fat trimmings.

The estimated availability of beef for grinding is based on the number of each type of cattle slaughtered in that region (Table 5.1). In 1992, approximately 73 percent of all cattle were slaughtered in the West Central region. Based on average carcass weights and percent of carcass going to ground beef, about 64 percent of total domestic raw product for ground beef originates in the West Central region. Some regional variation does exist in the fat content of ground beef blend categories (Table 5.2). However, the variation in fat content and the pattern of marketed ground beef blend within a region does not appear to correlate with the types of slaughter in that region. The lack of apparent geographical patterns in retail and HRI ground beef formulation may be due to a combination of post-slaughter movement of raw products, price differentials, and consumer preferences.

Variation in the number of steers and heifers slaughtered vs. cows and bulls slaughtered during a year may affect the ground beef composition on a seasonal basis (Figure 5.5). In the summer months (May through September) of FY 1988 through FY 1992, fed cattle accounted for 81.4 percent of all cattle slaughtered. In the winter months (October through April), fed cattle made up 78.6 percent of all cattle slaughtered (USDA:FSIS). Thus, ground beef formulated in the winter may have a slightly larger domestic cow and bull component.

Based on the number of each cattle type slaughtered and the percentage of carcass that goes into boneless manufacturing-grade beef (BMB), more than 50 percent of total domestic ground beef in 1992 came from steers and heifers (Table 5.3). There was little change in the types of cattle used to produce ground beef between 1980 and 1992 (assuming that the percentage of carcass used in ground beef did not change substantially). In general, these percentages can vary slightly depending on the market. When the demand for a certain cut of meat is low (e.g. chucks), it may be more profitable to use that cut in ground beef (Personal communication, R. Mucklow, Western States Meat Association, August 1993).

As discussed previously, per capita disappearance of ground beef has increased since 1980. Since the U.S. population has also increased since 1980, the supply of ground beef must have increased over the last decade. Since the total of domestic cattle slaughter and imported BMB (see below) remained relatively steady during that period, the increased supply of ground beef must have been due to a combination of other factors. One of those factors was an increase in the dressed weight of cattle, from 643 pounds in 1980 to 703 pounds in 1991 (USDA:NASS, b). However, the increase in carcass weight appears insufficient to entirely account for the increased ground beef supply[8]. Another factor was an increase in the amount of carcass going into ground beef, indirectly shown by a decrease in the consumption of choice beef cuts. A recent survey of packers indicated that the trend over the past 10 years was toward larger percentages of cow and bull carcasses becoming BMB (USDA:APHIS, 1994).

What is the availability of imported manufacturing-grade beef?

Importers of manufacturing-grade beef indicate that the product is widely distributed in U.S. markets. Processors mix the frozen imported beef with fresh domestic trimmings in order to keep the final ground beef product at temperatures low enough to inhibit bacterial growth. Guarantees of advertised leanness to within one-half of one percent, as opposed to the two percent tolerance applied to domestic product, also make the imported beef attractive to U.S. ground beef processors (Personal communications: T. Tobin, Butler Beef, September 1993; B. Chudy, Louis Dreyfus, September 1993; S. Orten, Monfort Inc., September 1993).

Imports into the U.S. account for almost one-half of the world trade in fresh, chilled, and frozen beef, and such imports make up over 90 percent of total U.S. beef imports (USDA:FAS, 1992). Approximately 15 percent of the fresh, chilled, and frozen beef and veal consumed in the U.S. originates from outside the country. Overall U.S. imports of fresh, chilled, and frozen beef increased slightly from the early 1980's into the 1990's (Figure 5.6). The annual average was 634,000 tons (USDA:ERS, b). Ninety percent of such imports originate from Australia, New Zealand, or Canada. At least 75 percent of the U.S. imports are a lean, manufacturing-grade product.

Because the U.S. Meat Import Law limits imports of fresh, chilled and frozen beef, veal, mutton, and goat meat into the U.S., significant increases in imports to the U.S. are not expected. Even Mexico's future exemption from the law's provisions under the North American Free Trade Agreement is not expected to increase imports into the U.S. (USDA:OE, 1992). If imports exceed the limit, quotas may be allocated to exporting countries based on historical trade patterns. However, the U.S. has adopted a policy of negotiating voluntary export restraint agreements with the exporting countries when it appears that the limit will be exceeded. Imports into the U.S.

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[8] Estimate based on number of head slaughtered and average dressed weights for 1980 and 1991, assuming a constant percentage of the carcass going into ground beef.

exceeding the agreed quantities are placed in bonded warehouses and held for release the following January. This arrangement leads to a concentration of imports in the early months of the calendar year and in the third quarter of the year (Hahn et al., 1990).

Approximately one-half of Australian beef exports are destined for the United States. Seventy percent of these exports are in the form of lean BMB used for hamburger or sausage products. Cattle raised on grass to heavier weights in the northern regions of Australia account for most of the boneless beef produced for export, with slaughter concentrated in April, May, and June (Dyck et al., 1989). Slaughterhouses tend to specialize for either the domestic or the international market. Australia's dependence on beef export markets -- approximately 75 percent of its domestic production is exported -- has led the export slaughterhouses to develop the world's most advanced technology for production of chilled beef and concurrently to set extremely high hygienic standards (*Meat and Poultry*, September 1990, pg. 22-30).

Approximately three-quarters of New Zealand's beef exports go to the United States, with shipments over the past 5 years averaging 210,000 tons annually, or approximately one-third of U.S. imports (USDA:ERS, b). This trade developed during the 1950's when declines in the U.S. dairy herd reduced supplies of domestically produced manufacturing-grade beef. Because half of New Zealand's beef production comes from dairy herds and because New Zealand's comparative advantage is in grass- rather than grain-fed livestock, the beef available for export is primarily lean manufacturing-grade beef. In recent years, a small industry has even developed raising bulls for production of very lean beef for export (Dyck et al., 1989). Slaughter typically commences in October and runs through January. As was true for Australia, New Zealand's dependence on export markets has led to extremely high hygienic standards for slaughterhouses where both USDA and European Community standards are enforced (Personal communication, M. Muirhead, NZ Meat Products Board, 1993).

Ninety percent of Canadian beef exports are destined for the U.S. Virtually all of the beef entering from Canada is manufacturing quality. The structure of the Canadian feeding and packing industry, as well as government freight subsidies for grain and oilseed exports, place it at a competitive disadvantage with the U.S. industry. Although Canada is not expected to expand its beef exports significantly in the future, imports to the U.S. did increase from 1980 to 1992 (Dyck et al., 1989). During the 5 years prior to Canada's exemption from the Meat Import Law due to the U.S.-Canadian Free Trade Agreement, fresh, chilled, and frozen beef exports to the U.S. averaged 75,000 tons annually and were relatively stable (Figure 5.6). From 1989 through 1991, the first 3 years following the exemption, exports were slightly higher, averaging 82,700 tons annually. Due to a reduction in government support to the dairy industry, Canadian cow slaughter jumped in 1992, and beef exports to the U.S. increased to 126,000 tons (USDA:ERS, b). The 1992 increase is seen as a 1-year event that is not expected to be repeated.

The remaining countries exporting fresh, chilled, and frozen beef to the U.S. are concentrated in Latin America and provide slightly less than 10 percent of imports on average. Costa Rica, Honduras, and the Dominican Republic account for approximately 70 percent of the U.S. imports coming from countries other than Australia, New Zealand, or Canada (Figure 5.6). These exports were very stable throughout the 1980's and into the early 1990's. In 1992, shipments to the U.S. fell slightly due to increased competition for pasture from crop production and increasing domestic beef consumption in the Central American countries (USDA:FAS, 1992).

How is ground beef marketed?

Once cattle have been slaughtered, ground beef production can flow through a variety of processing channels (Figure 5.7). Ground beef can be produced at different points along the marketing chain, including at slaughter plants, grinders, and retail outlets. There are currently 2,965 facilities that grind meat in the U.S., of which less than 900 slaughter cattle. Most retailers will also produce ground beef using purchased beef products. In 1992, there were 30,700 supermarkets with in-house meat departments.

A survey conducted in 1991 by Cattle-Fax investigated ground beef marketing by packers, HRI, grinders, and retailers (Cattle-Fax, 1991). Low response rates led to small sample sizes for the respective groups and fast food restaurants were overrepresented in the HRI group. In addition, grinders that also slaughtered were considered to be packers in one section of the survey, while the HRI section of the survey defined grinders to include both those that slaughtered and those that did not slaughter cattle. However, the information contained in the survey summary is the most comprehensive available.

According to the Cattle-Fax survey, 6.75 billion pounds of BMB were used to make ground beef in 1990. Only 15 percent of the material for retail ground beef is purchased from grinders that do not slaughter, while 24 percent and 38 percent originates directly from cow and fed beef packers, respectively. Retail stores primarily buy coarse ground (72%) or case-ready chubs (17.4%) (Cattle-Fax, 1991). Retail stores also purchase boxed beef (primal and sub-primal cuts of fed beef vacuum-packed into boxes) and do in-house fabrication, resulting in trimmings available for ground beef. The source of over 22 percent of retail ground beef is in-house trimmings, as more than half of retailers do some blending in the store.

About 59 percent of U.S. ground beef is purchased by HRI (Cattle-Fax, 1991). HRI purchase 98 percent of their ground beef from grinders and grinder/slaughterers, primarily in the form of patties (Figure 5.8) (Neel et al., 1994). This indicates that HRI ground beef undergoes an extra processing step as compared to retail ground beef. Grinders acquire 58 percent of their material from cow packers, 35 percent from fed beef packers, and almost 6 percent is imported lean BMB, which reflects the exclusion of grinders that do their own slaughtering. Most of the ground beef produced by grinders is sold to fast food firms (69.6%), while lesser amounts go to retailers (14.4%), restaurants (6.3%), and wholesale distributors (4.5%).

Almost all ground beef blends sold by HRI are in the 75-to-84 percent lean range (Figure 5.9). Lean content of ground beef sold by retail stores varies more widely, with almost one-third of the product below 75 percent lean and over 15 percent of the product above 84 percent lean. However, the weighted average lean content of retail and HRI ground beef is essentially the same (HRI = 80%, retail = 79% lean). Other studies of lean content of retail and HRI ground beef support the results of the Cattle-Fax survey (Johnson et al., 1994; Savell et al., 1991).

What has changed in the production of retail ground beef?

Purchasing of ground beef primarily as coarse ground and case-ready chubs by retailers appears to be a relatively new development. Historically, carcasses were purchased for fabrication on site at relatively small retail outlets where the trimmings would be converted to fresh ground beef. The introduction of boxed beef in the 1970's led to carcass fabrication moving from the local retail outlet to large retailers and wholesalers. Then, during the 1980's, the number of carcasses used by retailers and affiliated wholesalers to make boxed beef dropped from almost 1.7 million in 1982 to 0.33 million in 1991 as packers began dominating production of boxed beef (Personal

communication, G. Grinnell, USDA:P&SA, August 1993). By marketing boxed beef, packers can handle larger volumes, more efficiently use trimmings and byproducts, ship product that is easier to handle and takes less space, and respond to requests by retailers for specific primal or sub-primal cuts (Crom, 1988). In 1980, about 12 million fed steers and heifers went into boxed beef at slaughter plants. By 1990, this had increased to about 22.3 million head (USDA:P&SA).

Large retailers have moved to a system where they purchase ground beef regionally, usually under an agreement with one or two packers, according to demand for the product. Large chains use this regional approach to enable them to have a purchasing system which rapidly responds to changes in high volume sales of ground beef and to take advantage of cost efficiencies and productivity increases achieved when the processing is done at the packer level. Smaller chains can either rely on wholesalers or they can purchase boxed beef and create their own trimmings for making ground beef. The overall trend has been toward a centralization of ground beef production at the packer level and less handling of raw material at the retail level.

Based on the production channels for ground beef, is there any type of cattle which can be associated with O157 contamination?

It is not currently possible to focus on a specific type of cattle or marketing channel that might be associated with the contamination of ground beef by O157. Any type of cattle may be incorporated into ground beef. Dairy cattle, which have been the focus of much investigation related to human cases of O157, are no more likely than fed steers or heifers to constitute the largest proportion of any given pound of ground beef.

Table 5.1. Regional Availability of Beef for Grinding - 1992

Region*	Percent (%)				
	Steers	Cows	Heifers	Bulls	Total
West	27	53	11	9	100
West Central	48	20	26	6	100
East Central	38	38	17	7	100
North Central	20	62	4	14	100
Northeast	19	67	3	11	100
Southeast	3	76	3	18	100

* Regions:
West = WA, OR, ID, NV, CA
West Central = MT, WY, UT, AZ, NM, TX, OK, CO, ND, SD, NE, KS, IA, MO
East Central = MI, IL, IN, OH, KY
North Central = MN, WI
Northeast = ME, VT, NH, MA, RI, CT, NY, PA, NJ, DE, MD, VA, WV
Southeast = AR, LA, MS, AL, GA, FL, TN, NC, SC

Table 5.2. Geographic Distribution of Ground Beef Type and Percentage of Extractable Fat in Retail Ground Beef

Region	Ground Beef Type					
	Percent Marketed in Region			Fat Content Percent		
	Regular	Lean	Extra Lean	Regular	Lean	Extra Lean
Northeast	40.92	37.08	21.99	22.39	18.40	15.14
Southeast	34.65	51.76	13.59	23.76	19.92	16.10
North Central	21.10	37.83	41.07	26.16	22.16	18.80
South Central	42.49	34.56	22.94	23.59	18.26	16.38
Mountain	68.03	22.52	9.45	22.27	15.78	10.44
West	36.96	37.11	25.93	26.13	19.07	13.19
Overall Average for Retail	37.19	40.29	22.51	23.60	19.22	15.42
Overall Average for Purveyors (HRI)	40.9	40.5	5.9	20.20	17.45	12.43

Sources: Savell et al.,1991; Johnson et al.,1994

Table 5.3. Estimated* Percentage Composition of Total Domestic Ground Beef by Cattle Type, 1980 vs. 1992

Cattle Type	1980 (%)	1992 (%)
Steers	38.0	39.1
Cows	36.0	34.0
Heifers	18.2	19.2
Bulls	7.8	7.7

* Estimates based on data from USDA:NASS(b) and Cattle-Fax, 1991

Figure 5.1

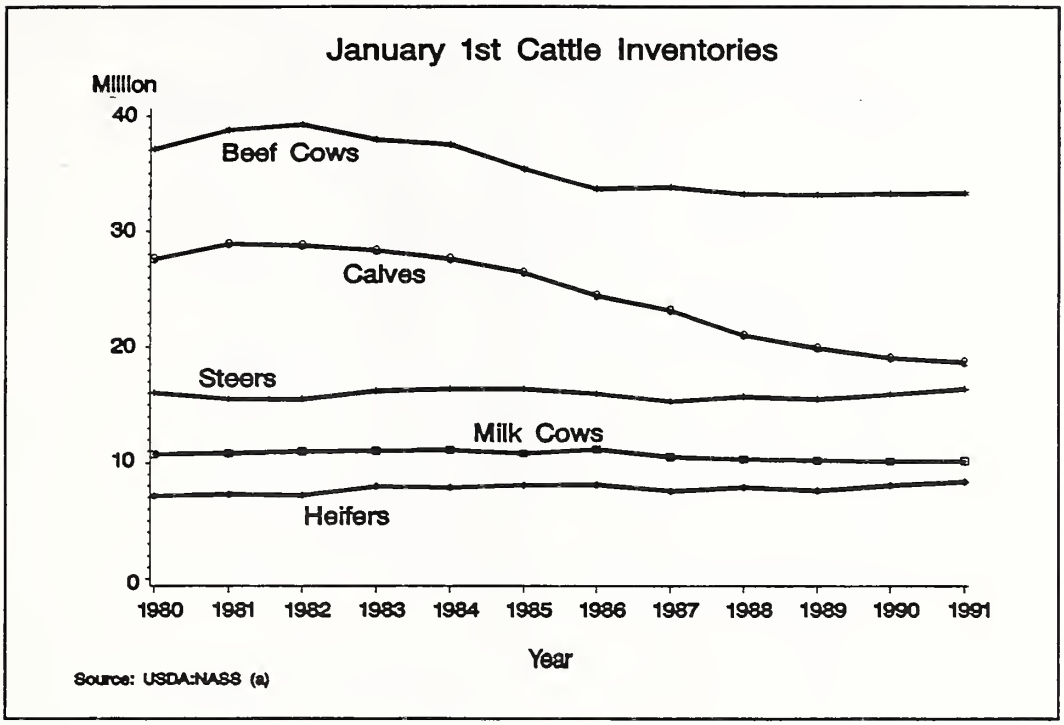


Figure 5.2

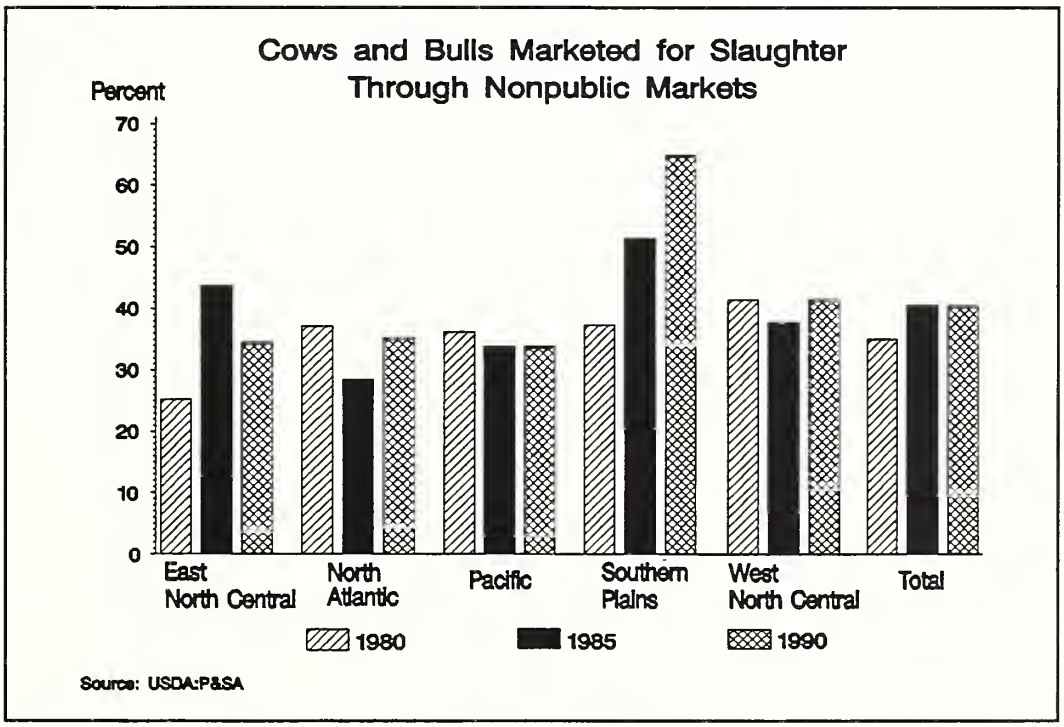
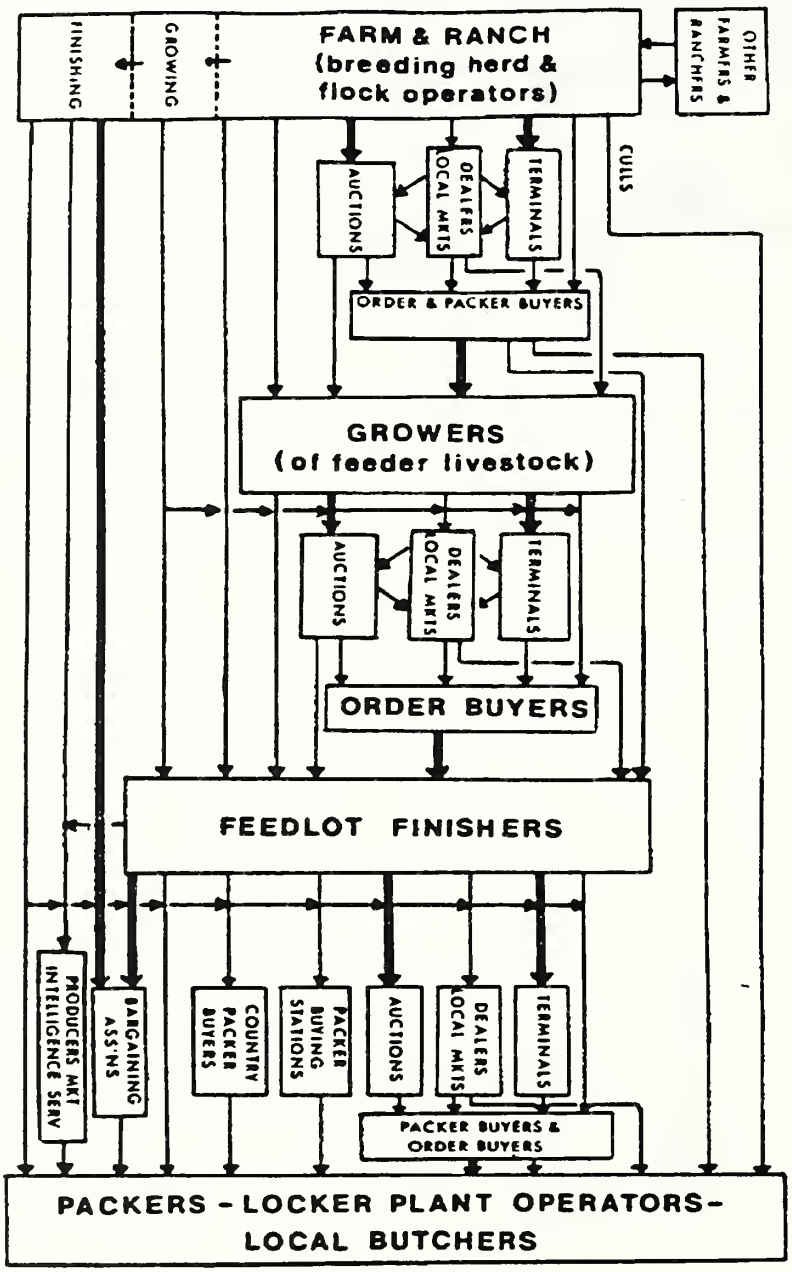


Figure 5.3

Livestock Marketing Channels



Source: McCoy and Sarhan, 1988

Figure 5.4

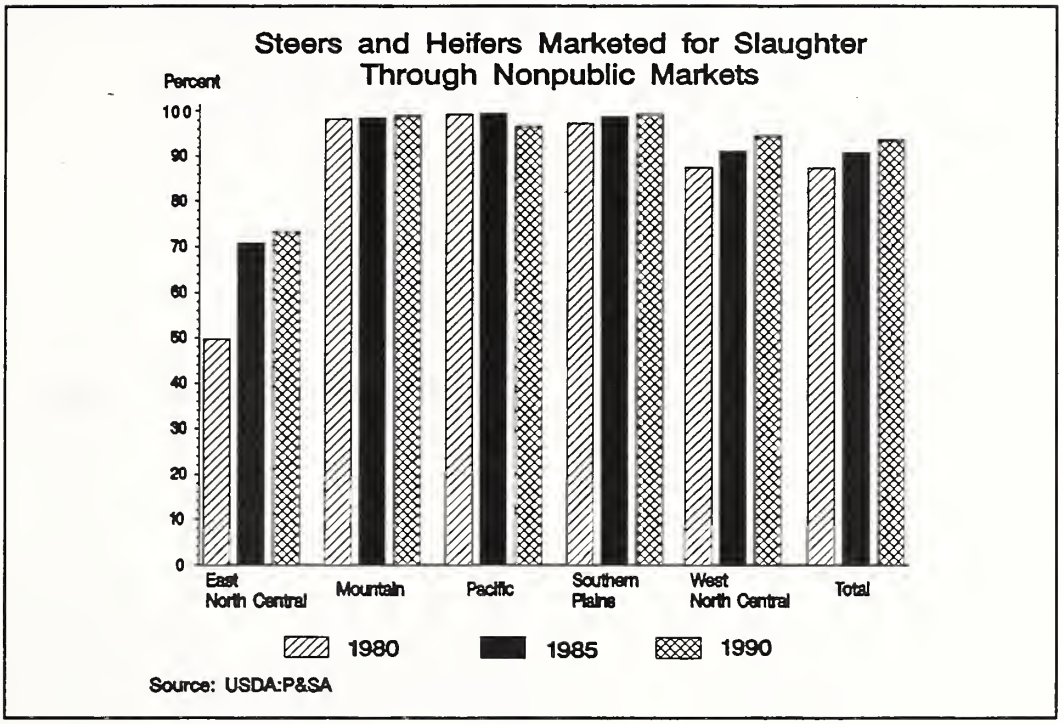


Figure 5.5

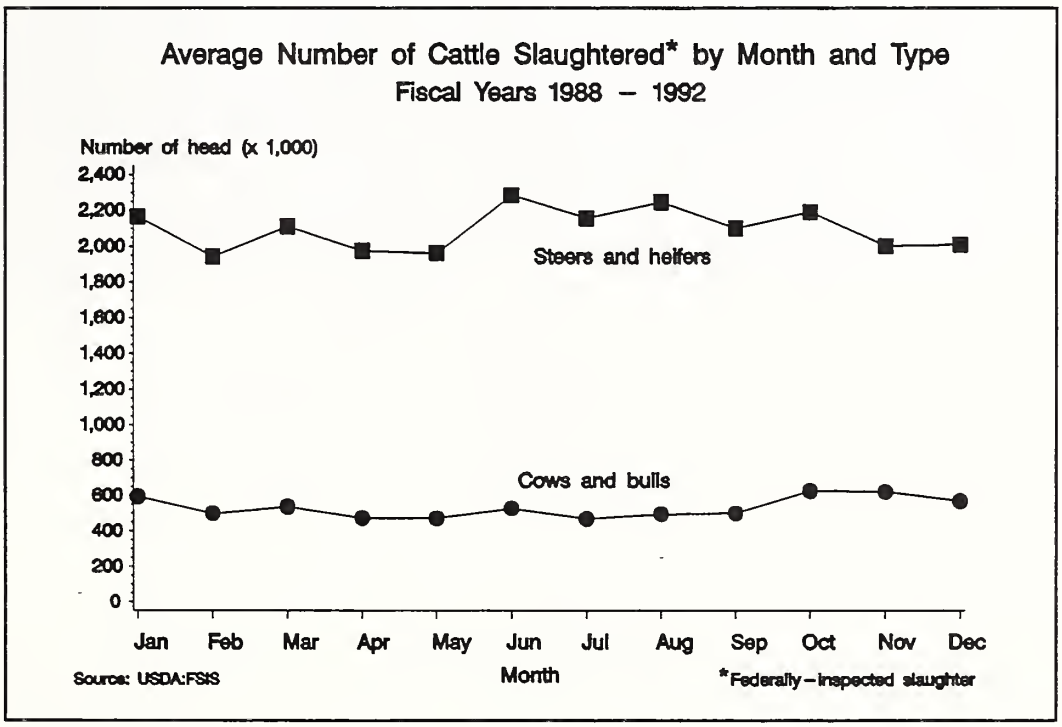
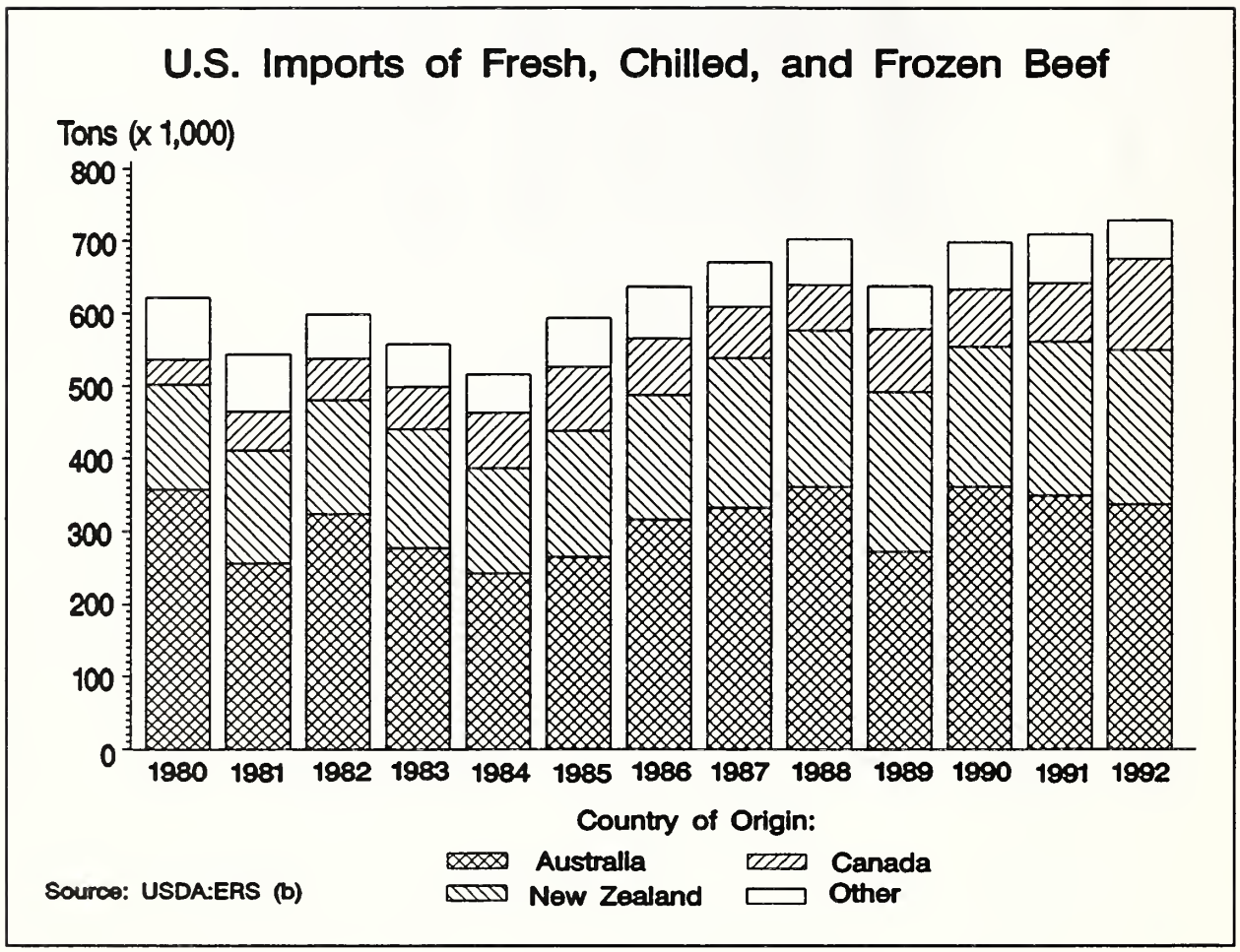
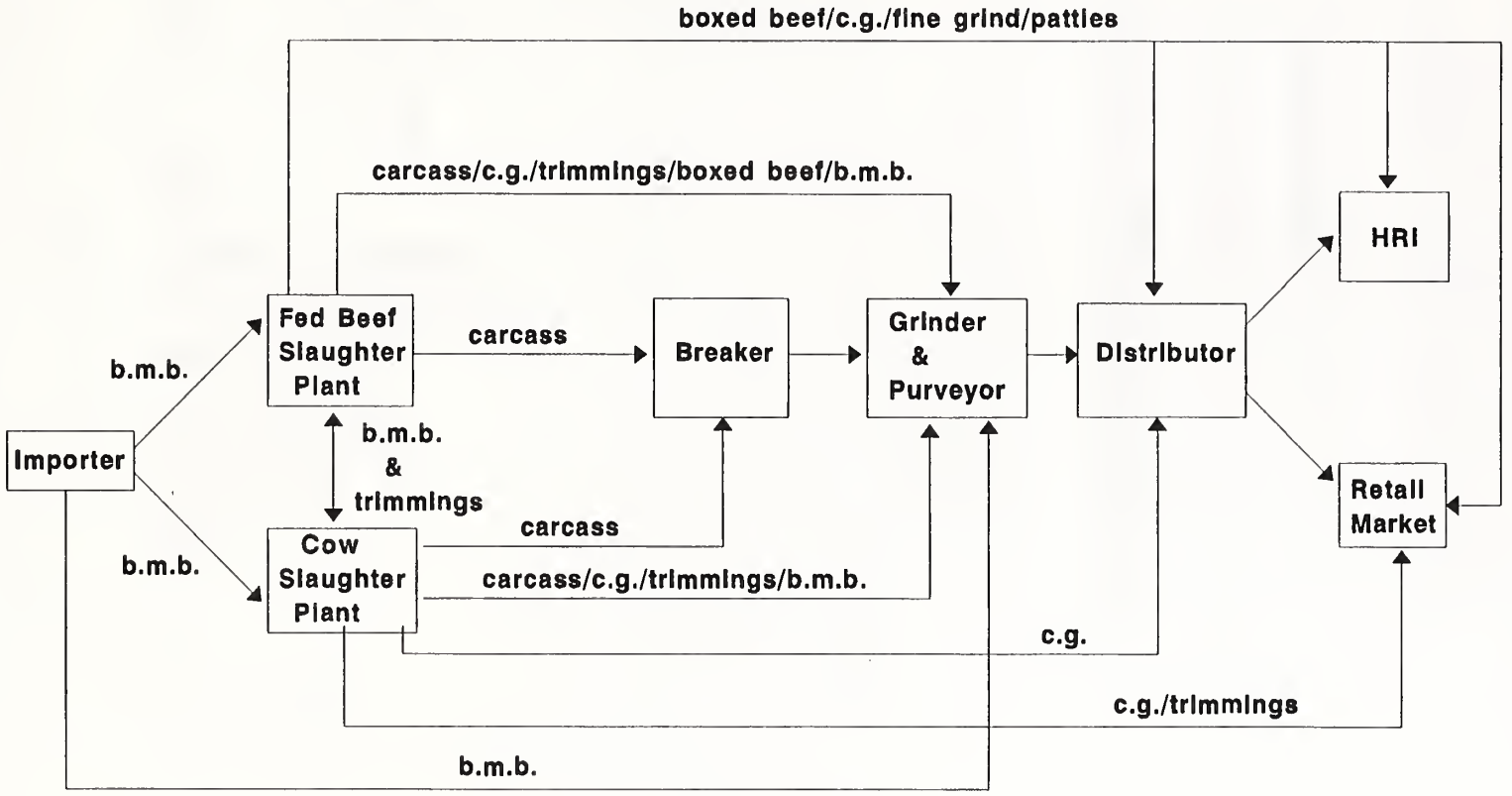


Figure 5.6


U.S. Imports of Fresh, Chilled, and Frozen Beef
Tons (x 1,000)
800
700
600
500
400
300
200
100
0
1980
1981
1982
1983
1984
1985
1986
1987
1988
1989
1990
1991
1992
Country of Origin:
Australia
Canada
New Zealand
Other
Source: USDA:ERS (b)

Possible Ground Beef Production and Distribution Channels



c.g.: coarse grind
b.m.b.: boneless manufacturing beef
HRI: Hotels, Restaurants, & Institutions

Figure 5.7

Figure 5.8

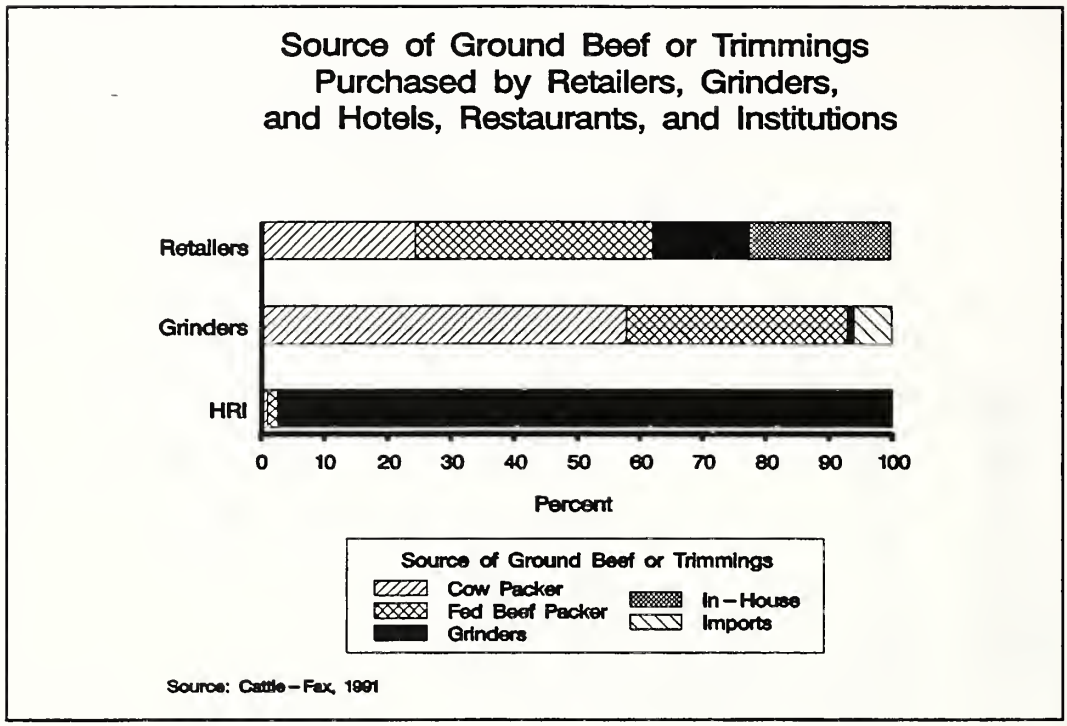
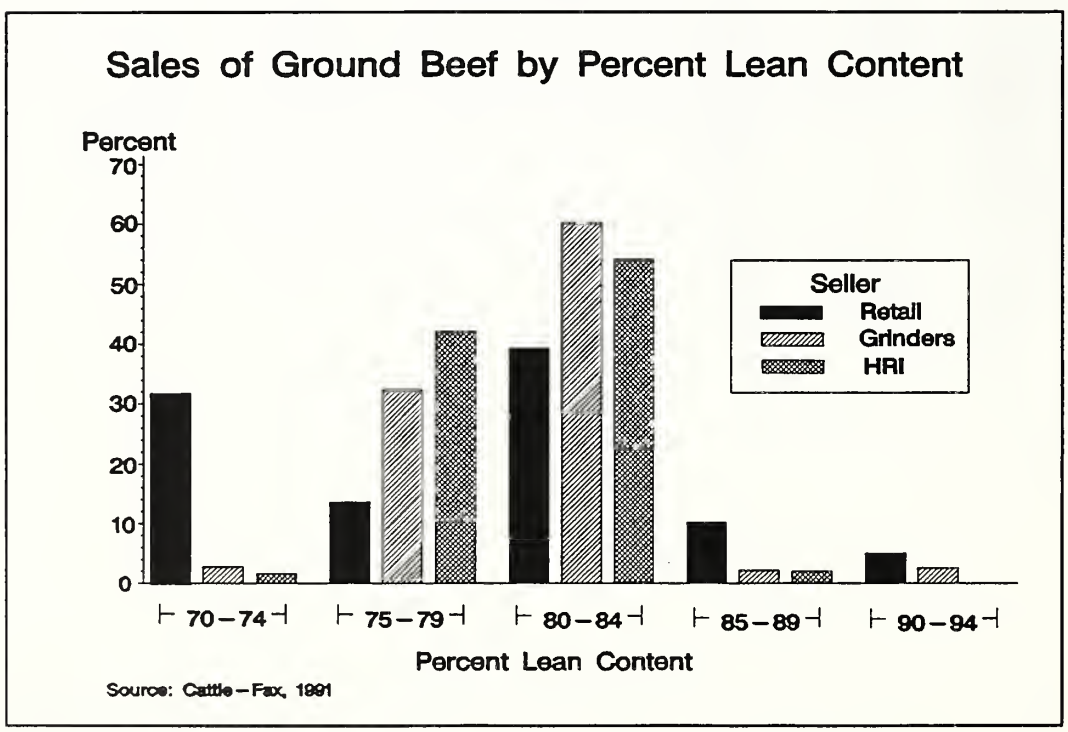


Figure 5.9



Intervention, Prevention, and Control of *E. coli* O157:H7

Can we eradicate O157 from U.S. farms, or only manage it?

Currently, there are no preharvest interventions known to control O157 and there is no evidence to suggest that O157 can be eliminated at the preharvest level in the immediate future. O157 is only one of many types of *E. coli* about which little ecologic information is known. The organism is widespread in the U.S. and can be found in many countries and on most continents. O157 can be found in both dairy and beef cattle for which there are not established preharvest risk factors. Identification of the organism in cattle is difficult due to sporadic shedding and the lack of clinical disease. Finally, the prevalence and implications of O157 in other species has not been actively pursued. Additional studies geared at understanding the ecology of the organism, enhancing detection, and identifying epidemiologic risk factors, especially at the preharvest level, are warranted. However, an immediate solution to allow eradication at the farm level is not likely.

The overall risk of illness from O157 is a function of the presence of the organism within a multifaceted animal production, ground beef manufacturing, and preparation process. The risk of O157 illness cannot be separated from the production to consumption continuum. The feasibility of eliminating that risk by focusing on a single stage in the continuum has yet to be established.

Understanding the risk of O157 and taking steps to manage it becomes a critical component of reducing human illness. This entails a better understanding of risk and changes to risk along the entire production to consumption continuum. Specific questions include: 1) how pre-slaughter activities of transportation, handling, and feeding affects shedding of the organism; 2) how different slaughter methods affect the amount of O157 contamination; 3) how the number of steps involved in the production of ground beef affects the risk of contamination; 4) what is the volume of ground beef that flows through the different production systems; 5) are there predominant channels with varying levels of risk for different types of cattle; 6) how do consumption patterns for those groups at highest risk relate to production and distribution patterns; and 7) are there specific production or distribution initiatives that target the most susceptible age groups and reduce overall risk? Specific knowledge throughout the entire system could then be used to identify varying levels of risk from O157 contamination. This knowledge would provide the basis to identify intervention options at critical points and better manage the overall risk of O157 contamination and subsequent illness.

What is HACCP and how does it apply to food safety?

The Hazard Analysis and Critical Control Point (HACCP) system shifts the primary emphasis for food safety away from end-product testing. Due to the heterogeneous distribution and low prevalence of O157 contamination, end-product testing, based on a statistically derived number of samples, is neither an effective nor a feasible means of assuring food safety. By contrast, HACCP focuses attention on the effective control of critical points in the production, slaughter, processing, storage, distribution, and final food preparation stages that could lead to unacceptable levels of contamination, cross-contamination, survival, or growth of foodborne pathogens and other microorganisms.

A specific HACCP program should eventually be developed and implemented for each individual farm, slaughter facility, grinder, or other component of a food production and consumption continuum. The development of a HACCP plan consists of seven basic principles. These principles include: 1) conducting a hazard analysis; 2) identifying the critical control points (CCP) required to

control an identified hazard; 3) establishing critical limits for each CCP; 4) establishing CCP monitoring requirements; 5) establishing corrective actions to be taken when monitoring indicates that there is a deviation from a critical limit; 6) establishing effective record keeping procedures and documentation; and 7) establishing procedures for verifying that the HACCP plan is working effectively. The intent is to ensure that any product leaving a given phase of production or a given location is as safe or safer than it was when it entered.

What are other countries doing to control O157 and other foodborne pathogens?

To date, no countries have instituted control measures specifically for O157. However, several countries have adopted programs to reduce the level of contamination from *Salmonella*. These programs, directed in particular at poultry and egg production, have included preharvest control measures.

For example, Swedish hatcheries participating in a voluntary program are required to follow strict hygiene and management procedures. Producers receive endemnity payments for flock destruction due to *Salmonella* infection only if they have received their chicks from the participating hatcheries. Should contamination occur on the farm, guidelines exist for cleaning and repopulating the premises, including the use of competitive exclusion (probiotic) cultures in two consecutive flocks following contamination. In addition to these requirements, controls have been placed on drug and feed use to reduce the risk of *Salmonella* contamination (Wierup, 1993).

An economic analysis of the Swedish program for 1992 indicated that the benefits of controlling *Salmonella* equaled or exceeded the costs. Cost estimates for the current levels of illness and control measures ranged from 112 to 118 million Swedish kronor, while costs of illness and control in the absence of the program were estimated at 117 to 266 million Swedish kronor (Engvall and Andersson, 1993).

What types of preharvest research are needed on O157 in the U.S.?

In order to fill the largest voids in our knowledge of O157 as quickly as possible, research should be targeted both at the farm and at preslaughter events. Sampling individual animals before leaving the farm, at the auction barn, in the feedlot, and immediately prior to slaughter may add to our understanding of the onset and duration of fecal shedding. Emphasis should be placed on the cleanliness of hides and haircoats prior to slaughter in order to determine the importance of this factor in O157 contamination.

On-farm research should investigate the ecology of O157. Additional information on shedding patterns could be obtained through long-term fecal sampling of O157-positive herds. The effect of cattle stressors, such as changes in diet and transportation, on gastrointestinal microflora is relatively unknown. Environmental sampling including feed, water, soil, and other livestock could provide valuable information on O157 survivability and maintenance in a herd, as well as modes of transmission. Results from a limited number of studies were inconclusive on the effect of various management practices, such as ionophore use, on O157 prevalence. Studies to identify O157 risk factors should be a high priority. Information gained from such work could potentially also apply to the control of other foodborne pathogens. Research efforts should include both dairy and beef animals.

What is competitive exclusion and is it a promising control option?

Competitive exclusion is the use of a preparation of viable, nonpathogenic microorganisms (a probiotic) to exclude or inhibit undesirable microorganisms in the gastrointestinal tract of a host animal. There are currently no probiotics marketed in the U.S. which have approved prophylactic or therapeutic uses against O157 or other organisms in cattle. The extent of probiotic usage in cattle production is unknown, but a recent survey revealed that 5.2 percent of respondents in the dairy industry used probiotic feed supplements (Hoard's Dairyman, 1992).

The use of competitive exclusion is advocated primarily for animals undergoing feed changes, transportation, crowding, or other stress. Stressed animals often develop an imbalance in the intestinal microflora in which the minor species of coliforms, including *E. coli*, overgrow and the major species of the normal anaerobic flora are inhibited. Such stress and overgrowth could conceivably lead to the shedding of organisms, such as O157, that otherwise might not have been present in large numbers.

The efficacy of probiotics in various species has been variable (Rolfe, 1991; Scheuermann, 1993; Stavric, 1992; Gill et al., 1987; Higginbotham and Bath, 1991; Montes and Pugh, 1993). Attempts to establish a probiotic flora in the gastrointestinal tract of cattle have had mixed results. Disparate results were obtained in published studies on probiotics in calves and feedlot cattle (Gill et al., 1987; Higginbotham and Bath, 1993; Montes and Pugh, 1993). Despite differences in the gastrointestinal tract physiology and microflora of monogastrics and of ruminants, studies in poultry may suggest potential strategies for the development of a probiotic effective against O157 in cattle. Protection of chicks from colonization of O157 has been documented using a culture of undefined species composition isolated from feces of specific pathogen-free adult birds (Stavric et al., 1992). Two probiotic products for competitive exclusion of human enteropathogens in poultry appear likely to be approved for commercial use.

Bacterial strains which offer potential for further development of a therapeutic probiotic in cattle have been identified. A mixed culture of two bacterial strains, which are normal colonizers of the intestinal tract of humans and most other animals, inhibited *Salmonella typhimurium* and O157 *in vitro* (Hinton et al., 1991). Inhibition of O157 was also demonstrated *in vitro* for a proportion of the *Lactobacillus* strains in two probiotics developed outside the U.S. (Chateau et al., 1993).

The development of a prophylactic or therapeutic probiotic targeting O157 in cattle is likely to involve considerable long-term research and require a broad spectrum of bacterial genera which normally occupy niches in the gastrointestinal tract of healthy cattle. Thus, competitive exclusion is not a solution to the O157 problem that will be available in the near future.

Would rapid tests be useful?

Currently, efforts are underway to develop rapid testing methods for detecting the presence of O157 in cattle. Some methods under investigation include an adenosine triphosphatase (ATP-ase) test, a serologic antigen test, and a polymerase chain reaction (PCR) test. Rapid tests might be used at various points along the farm-to-consumer continuum including on-farm, prior to slaughter, on carcasses, on slaughter facility equipment and on finished product. Rapid tests that have a high sensitivity and specificity would probably be practical in applications such as the monitoring of slaughter equipment.

The application of a rapid test to screen for the presence of O157 is probably not practical because of the overall low prevalence of O157 found in cattle to date, the transient shedding of the

organism from individual cattle and the low counts of O157 that have been detected in ground beef. For example, if 1 percent of all cattle were truly positive for O157 and all 30 million cattle slaughtered in a year were tested with a 99 percent sensitive and specific test, about 3,000 positive animals would be missed (false-negatives) and 297,000 animals would be misclassified as being O157-positive (false-positives). The costs associated with 30 million tests per year would be excessive, especially in light of the number of false-positive and false-negative animals which would result.

Are organic sprays or washes effective against O157?

At the slaughter level, organic sprays and washes have been shown to reduce microbial counts on carcass surfaces. However, based on a recent study, O157 appears to be more resistant to organic acid decontamination than other foodborne pathogens, including *Salmonella* (Siragusa and Nettles, 1993). Although, under certain conditions, acetic and lactic acid washes have been found to reduce levels of O157, they did not result in reductions that would render the carcass microbially safe. Preliminary findings from a current study indicate that ozonated water and hydrogen peroxide washes may be a better means of decontaminating carcass surfaces (Personal communication, B. Morgan, Colorado State University, February 1994).

Is irradiation of ground beef an effective and practical intervention?

Irradiation is the only currently known method, aside from cooking, by which pathogens can be eliminated from food. Over 40 years of research in the U.S. and other countries has shown that irradiation of food is safe and effective (Jones, 1992). The FDA approved irradiation for potatoes and grains as early as the 1960's. More recently, the process was approved for raw pork (1985), fresh fruit (1986), and raw poultry (1990). The approved dose is low enough to ensure that irradiated food will spoil before it can become toxic. Nutrient losses from irradiation are in the same range or lower than those caused by other commercial processing methods. The formation of minute quantities of substances not present before irradiation has been documented, but such substances pose no known health hazard to the consumer. Current proponents of beef irradiation include the National Food Processors Association, the American Medical Association, the World Health Organization, and the American Meat Institute (*Wall Street Journal*, February 4, 1994, pg. B1).

Four sources of energy exist that can provide the appropriate dose for irradiation of food: two isotopes, cobalt-60 and cesium-137, and two types of particle accelerators. The particle accelerators yield no environmental waste; however, they are technically more complex and their usage is limited because they kill only surface bacteria. Irradiation with cobalt-60 is currently used to sterilize disposable medical devices in the U.S. When cobalt-60 can no longer be used for such purposes, it can be used as a supplemental source for food irradiation and thereby decrease environmental waste.

Although approved for some food products, irradiation has not been widely implemented by food processors primarily because it requires a major financial investment. The cost-effectiveness of such an investment is difficult to assess because the degree of consumer acceptance is unpredictable. However, it appears that acceptance may be largely a matter of education. In South Africa, 90 percent of consumers react positively to irradiation of food (Jones, 1992). In the Netherlands, consumers' attitudes changed when they recognized that irradiation decreases the use of pesticides and other chemicals (Jones, 1992).

The primary questions concerning irradiation relate to large scale feasibility, such as would be necessary for ground beef. The volume of ground beef produced in the U.S. is too large to irradiate given the present infrastructure, as there is currently only one commercial food irradiation facility in the U.S. In addition, although cobalt-60 from medical uses could be utilized, extensive ground beef irradiation would necessitate the production and eventual disposal of additional isotope material.

Although the use of irradiation in ground beef has promise and should be pursued, it does not provide a short-term solution to the O157 problem. Any facility irradiating meat or poultry would require the approval of the Food Safety and Inspection Service, the Environmental Protection Agency, the Nuclear Regulatory Commission, the Occupational Safety and Health Administration, the Food and Drug Administration, and perhaps state licensing boards (Personal communication, D. Englejohn, USDA:FSIS:PPID, October 1993).

Does tracing O157 cases back to the farm of origin have value?

Tracebacks have been proposed as an important component of a food safety agenda. In the case of O157, tracebacks could potentially provide valuable information about on-farm factors and production processes associated with strains of O157 known to have caused human disease. In addition, farms or ranches traced back from human cases of O157 would be the best premises on which to study the ecology of the organism. From an immediate disease prevention perspective, however, tracebacks are currently of uncertain value because so little is yet known about on-farm factors which may be related to control or management of the organism.

Tracebacks involving ground beef would be especially difficult to carry out to the farm level with a high degree of precision. The complexity of marketing channels, as well as the variety of sources that combine to make up ground beef, have already led to problems in epidemiologic tracebacks following O157 outbreaks. Identification of the specific animal and originating farm that may have been the source of contamination is virtually impossible. It is conceivable, perhaps likely, that parts of over 100 carcasses could go into any given 2,000 pound "combo bin" of trimmings and lean used to produce ground beef. Thus, even with a highly dependable system of individual animal identification, the determination of individual animal contributions to any given pound of product is unlikely.

Even if it were possible to accurately trace a given pound of product to its source animal(s) and then to its source premises, there would be no certainty that the O157 which contaminated that product was ever associated with that (those) premises. Contamination of animals or product might occur at any point along the production continuum. Since the exact mechanism(s) of contamination has not yet been elucidated, we may know which product the O157 was in, but not where the O157 contamination of that product occurred. Contamination of a given product may occur from an adjacent carcass, contaminated equipment used during slaughter, or from dozens of other sources which might not have any relationship to the product's origin. Thus, tracebacks may be more valuable in determining patterns of production that are common to contaminated product, if such patterns exist, than in finding premises on which disease-causing O157 exist.

References

Albert, M.J.; Bhat, P.; Rajan, D. et al. 1978. Jejunal microbial flora of southern Indian infants in health and with acute gastroenteritis. J Med Microbiol 11:433-440.

AMI. 1992. 1992 Meat and poultry facts. American Meat Institute, Washington, DC.

Anderson, M.A. and Horn, G.W. 1987. The effect of lasalocid on weight gains, ruminal fermentation and forage intake of stocker cattle grazing winter wheat pasture. J Anim Sci 65:865-871.

Barrow, P.A.; Fuller, R. and Newport, M.J. 1977. Changes in the microflora and physiology of the anterior intestinal tract of pigs weaned at 2 days, with special reference to the pathogenesis of diarrhea. Infect Immun 18:586-595.

Beery, J.T.; Doyle, M.P. and Schoeni, J.L. 1985. Colonization of chicken cecae by *Escherichia coli* associated with hemorrhagic colitis. Appl Environ Microbiol 49:310-315.

Belongia, E.A.; MacDonald, K.L.; Parham, G.L. et al. 1991. An outbreak of *Escherichia coli* 0157:H7 colitis associated with consumption of precooked meat patties. J Infect Dis 164:338-43.

Belongia, E.A; Osterholm, M.T.; Soler, J.T. et al. 1993. Transmission of *Escherichia coli* 0157:H7 infection in Minnesota child day-care facilities. J Am Med Assoc 269:883-888.

Bergen, W.G. and Bates, B.B. 1984. Ionophores: their effect on production efficiency and mode of action. J Anim Sci 58:1465-1483.

Bettelheim, K.A.; Brown, J.E.; Lolekha, S. et al. 1990. Serotypes of *Escherichia coli* that hybridized with DNA probes for genes encoding Shiga-like toxin I, Shiga-like toxin II, and serogroup 0157 enterohemorrhagic *E. coli* fimbriae isolated from adults with diarrhea in Thailand. J Clin Microbiol 28:293-295.

Bhan, M.K.; Raj, P.; Levine, M.M. et al. 1989. Enteroaggregative *Escherichia coli* associated with persistent diarrhea in a cohort of rural children in India. J Infect Dis 159:1061-1064.

Blake, P.A.; Cohen, M.L.; Davis, B.R. et al. 1983. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. N Engl J Med 308:681-685.

Borczyk, A.A.; Lior, H. and Thompson, S. 1989. Sorbitol negative *Escherichia coli* O157 other than H7. J Infect Dis 18:198-199.

Borczyk, A.A.; Karmali, M.A.; Lior, H. et al. 1987. Bovine reservoir for verotoxin-producing *Escherichia coli* O157:H7. Lancet 1:98.

Borczyk, A.A.; Harnett, N.; Lombos, M. et al. 1990. False-positive identification of *Escherichia coli* 0157 by commercial latex agglutination tests. Lancet 336:946-947.

Brownell, J.R.; Sadler, W.W. and Fanelli, M.J. 1969. Factors influencing the intestinal infection of chickens with *Salmonella typhimurium*. Avian Dis 13:804-816.

Browning, N.G.; Botha, J.R.; Sacho, H. et al. 1990. *Escherichia coli* 0157:H7 haemorrhagic colitis. Report of the first South African case. S Afr J Surg 28:28-29.

Bryan, F.L. 1983. Epidemiology of milk-borne diseases. J Food Protect 46:637-649.

Bryant, H.E.; Athar, M.A. and Pai, C.H. 1989. Risk factors for *Escherichia coli* 0157:H7 infection in an urban community. J Infect Dis 160:858-864.

Bulte, M. 1991. Significance and detection of *E. coli* O157:H7 and other verotoxigenic serovars in slaughter animals and in food of animal origin in the Federal Republic of Germany. Zentralbl Hyg Umweltmed 192:173.

Bureau of Foodservice Research. 1992. Restaurants and institutions national consumer survey, 1992 tastes of America. Cahners Publishing Company, Des Plaines, IL.

Byrne, B.M. and Dankert, J. 1979. Volatile fatty acids and aerobic flora in the gastrointestinal tract of mice under various conditions. Infect Immun 23:559-563.

Cahoon, F. and Thompson, J.S. 1987. Frequency of *Escherichia coli* 0157:H7 isolation from stool specimens. Can J Microbiol 33:914-915.

Caprioli, A.; Edefonti, A.; Bacchini, M. et al. 1990. Isolation in Italy of a verotoxin-producing strain of *Escherichia coli* 0157:H7 from a child with hemolytic-uremic syndrome. Eur J Epidemiol 6:102-104.

Carter, A.O.; Borczyk, A.A.; Carlson, J.A.K. et al. 1987. A severe outbreak of *Escherichia coli* 0157:H7 - associated hemorrhagic colitis in a nursing home. New Engl J Med 317:1496-1500.

Cattle-Fax. 1991. An economic analysis of low-fat ground beef. Presented to The National Cattlemen's Association and The Cattlemen's Beef Board by Cattle-Fax, Englewood, CO.

CDC. 1978. *Campylobacter* enteritis--Colorado. MMWR 27:226,231.

CDC. 1981a. Raw milk associated illness--Oregon, California. MMWR 30:90-92,97.

CDC. 1981b. Outbreak of *Campylobacter* enteritis associated with raw milk--Kansas. MMWR 30:218-220.

CDC. 1984. *Campylobacter* outbreak associated with certified raw milk products--California. MMWR 337:562.

CDC. 1993a. Preventing foodborne illness: *Escherichia coli* O157:H7. National Center for Infectious Diseases, Division of Bacterial and Mycotic Diseases, Atlanta, GA.

CDC. 1993b. Preliminary report: foodborne outbreak of *Escherichia coli* 0157:H7 infections from hamburgers - Western United States. MMWR 42:85-86.

Chapman, P.A.; Wright, D.J. and Norman, P. 1989. Verotoxin-producing *Escherichia coli* infections in Sheffield: cattle as a possible source. Epidemiol Infect 102:439-445.

Chapman, P.A.; Siddons, C.A.; Zadik, P.M. et al. 1991. An improved selective medium for the isolation of *Escherichia coli* 0157. J Med Microbiol 35:107-110.

Chart H.; Rowe B.; v.d. Kar, N. et al. 1991. Serological identification of *Escherichia coli* 0157 as cause of haemolytic uraemic syndrome in the Netherlands (letter). Lancet 337:437.

Chateau, N.; Castellanos, I. and Deschamps, A.M. 1993. Distribution of pathogen inhibition in the *Lactobacillus* isolates of a commercial probiotic consortium. J Applied Bact 74:36-40.

Christie, D.L.; Clausen, C.R.; Hickman, R.O. et al. 1990. *Escherichia coli* O157:H7 and the hemolytic uremic syndrome: importance of early cultures in establishing the etiology. J Infect Dis 162:553-556.

Cieslak, P.R.; Barrett, T.J.; Griffin, P.M. et al. 1993. *Escherichia coli* O157:H7 infection from a manured garden. Lancet 342:367.

Clarke, R.; McEwen, S. and Harnett, N. 1988. The prevalence of verotoxin-producing *Escherichia coli* (VTEC) in bovines at slaughter. Abstracts of the Annual Meeting of the American Society for Microbiology, Miami Beach, FL.

Cohen, R.; Kalser, M.H.; Arteaga, I. et al. 1967. Microbial intestinal flora in acute diarrheal disease. J Am Med Assoc 201:835-840.

Copperstock, M.S. and Zedd, A.J. 1983. Intestinal flora of infants. In: Hentges, D.J. (ed.). Human intestinal microflora in health and disease. Academic Press, New York, NY, pg. 79-99.

Cordovez, A.; Prado, V.; Maggi, L. et al. 1992. Enterohemorrhagic *Escherichia coli* associated with hemolytic-uremic syndrome in Chilean children. J Clin Microbiol 30:2153-2157.

Cravioto, A.; Reyes, R.E.; Trujillo, F. et al. 1990. Risk of diarrhea during the first year of life associated with initial and subsequent colonization by specific enteropathogens. Am J Epidemiol 131:886-904.

Crom, R.J. 1988. Economics of the U.S. meat industry. U.S. Dept. of Agriculture, Economic Research Service, Agricultural Information Bulletin No. 545, Washington, DC.

Crosse, B.A. and Naylor, J.R. 1990. Haemolytic-uraemic syndrome in adults. Lancet 30:1528-1529.

Cullor, J. 1993. Is there an *E.Coli* 0157:H7 in your practice? Presented at the annual meeting of the American Association of Bovine Practitioners, Albuquerque, NM, September 1993.

D'Aoust, J.-Y. 1989. Contemporary concerns on the microbiological safety of milk and dairy products. Modern microbiological methods for dairy products, International Dairy Federation special issue No. 8901:15-45.

D'Aoust, J.-Y.; Park, C.E.; Szabo, P.A. et al. 1988. Thermal inactivation of *Campylobacter* species, *Yersinia enterocolitica*, and hemorrhagic *Escherichia coli* O157:H7 in fluid milk. J Dairy Sci 71:3230-3236.

deGrace, M.; Laurin, M.F.; Belanger, C. et al. 1976. *Yersinia enterocolitica* gastroenteritis outbreak--Montreal. Can Dis Weekly Rep 2:41-44.

Dev, V.J.; Main, M. and Gould, I. 1991. Waterborne outbreak of *Escherichia coli* 0157. Lancet 337:1412.

Disease Surveillance. 1984. Kindergarten field trip to a farm, June 25, 1984, Vancouver Island. Disease Surveillance, British Columbia 5:201-203.

Dorn, C.R. 1988. Hemorrhagic colitis and hemolytic uremic syndrome caused by *Escherichia coli* in people consuming undercooked beef and unpasteurized milk. J Am Vet Med Assoc 193:1360.

Dorn, C.R. 1993. Review of foodborne outbreak of *Escherichia coli* O157:H7 infection in the western United States. J Am Vet Med Assoc 203:1583-1587.

Dorn, C.R. and Angrick, E.J. 1991. Serotype O157:H7 *Escherichia coli* from bovine and meat sources. J Clin Microbiol 29:1225-1231.

Doyle, M.P. and Schoeni, J.L. 1984. Survival and growth characteristics of *Escherichia coli* associated with hemorrhagic colitis. Appl Environ Microbiol 48:855-856.

Doyle, M.P. and Schoeni, J.L. 1987. Isolation of *Escherichia* coli 0157:H7 from retail fresh meats and poultry. Appl Environ Microbiol 53:2394-2396.

Dyck, J.; Goodloe, C. and Morgan, N. 1989. The Pacific beef industry and trade. U.S. Dept. of Agriculture, Economic Research Service, Agriculture and Trade Analysis Division, Pacific Rim Agriculture and Trade Report, RS-89-3, Washington, DC.

Engvall, A. and Andersson, Y. 1993. The economics of the Swedish *Salmonella* control: a cost/benefit analysis. Presented at the NVI/WHO International Course on *Salmonella* Control in Animal Production and Products, Malmo, Sweden.

Fallert, R.F.; Blayney, D.P. and Miller, J.J. 1990. Dairy: background for 1990 farm legislation. U.S. Dept. of Agriculture, Economic Research Service, Staff Report AGES9020.

Farmer, J.J.; Griffin, P.M.; Wells, J.G. et al. 1988. Diarrheal stool cultures that yield no apparent pathogen: *Escherichia coli* O157:H7. In: Gambino, S.R. (ed.). American Society of Clinical Pathologists Continuing Education Program. Centers for Disease Control, Atlanta, GA, pg. 1-5.

Farmer, J.J.; Potter, M.E.; Riley, L.W. et al. 1983. Animal models to study *Escherichia coli* O157:H7 isolated from patients with hemorrhagic colitis. Lancet 1:702-703.

Farrag, S.A. and Marth, E.H. 1992. *Escherichia coli* O157:H7, Yersinia enterocolitica and their control in milk by the lactoperoxidase system: a review. Lebensmittel-Wissenschaft und - Technologie 25:201-211.

FDA. 1993. Data from the raw milk intrastate survey. U.S. Dept. of Health and Human Services, Milk Safety Branch, Washington, DC.

Federal Veterinarian. 1993. Ten *E. coli* outbreaks. The National Association of Federal Veterinarians, Washington, DC 50(10):7.

Fischer, L.J.; Erfle, J.D. and Sauer, F.D. 1971. Preliminary evaluation of the addition of glucogenic material to the rations of lactating cows. Canadian J Anim Sci 51:721-727.

Fish, N.A.; Finlayson, M.C. and Carere, R.P. 1967. Salmonellosis: report of a human case following direct contact with infected cattle. Can Med Assoc J 96:1163-1165.

Francis, D.H.; Collins, J.E. and Duimstra, J.R. 1986. Infection of gnotobiotic piglets with an *Escherichia coli* O157:H7 strain associated with an outbreak on hemorrhagic colitis. Infect Immun 51:953-956.

Galton, M.M.; Smith, W.V.; McElrath, H.B. et al. 1954. *Salmonella* in swine, cattle, and the environment of abattoirs. J Infect Dis 95:236-245.

Garber, L.; Wells, S.; Hancock, D. et al. 1994. *E. coli* 0157:H7 in dairy heifer results of a case-control study (unpublished).

Gill, D.R.; Smith, R.A. and Ball, R.L. 1987. The effect of probiotic feeding on health and performance of newly-arrived stocker calves. Animal Science Research Report, Oklahoma Agricultural Experiment Station, pg. 202-204.

Giraldi, R.; Guth, B.E.C. and Trabulsi, L.R. 1990. Shiga-like toxin among *Escherichia coli* strains and other bacteria isolated from diarrhea in Sao Paulo, Brazil. J Clin Microbiol 28:1460-1462.

Glass, K.A.; Loeffelholz, J.M.; Ford, J.P. et al. 1992. Fate of *Escherichia coli* O157:H7 as affected by pH or sodium chloride and in fermented, dry sausage. Appl Environ Microbiol 58:2513-2516.

Gonzalez, E.A. and Blanco, J. 1989. Serotypes and antibiotic resistance of verotoxigenic (VTEC) and necrotizing (NTEC) *Escherichia coli* strains isolated from calves with diarrhoea. FEMS Microbiol Lett 60:31-36.

Gracey, M. and Stone, D.E. 1972. Small-intestinal microflora in Australian aboriginal children with chronic diarrhoea. Aust NZ J Med 3:215-219.

Gracey, M.; Sunoto, S. and Stone, D.E. 1973. Microbial contamination of the gut: another feature of malnutrition. Am J Clin Nutr 26:1170-1174.

Grau, F.H. and Smith, M.G. 1974. *Salmonella* contamination of sheep and mutton carcasses related to pre-slaughter holding conditions. J Appl Bact 37:111-116.

Griffin, P.M. and Tauxe, R.V. 1991. The epidemiology of infections caused by *Escherichia coli* 0157:H7, other enterohemorrhagic *E. coli*, and the associated hemolytic uremic syndrome. Epidemiol Rev 13:60-98.

Griffin, P.M. and Tauxe, R.V. 1993. *Escherichia coli* 0157:H7-human illness in North America, food vehicles and animal reservoirs. Int Food Safety News 2:15-17.

Griffin, P.M.; Olmstead, L.C. and Petras, R.E. 1990. *Escherichia coli* 0157:H7-associated colitis. Gastroenterol 99:142-149.

Griffin, P.M.; Ostroff, S.M.; Tauxe, M.D. et al. 1988. Illnesses associated with *Escherichia coli* 0157:H7 infections. A broad clinical spectrum. Ann Intern Med 109:705-712.

Hahn, W.F.; Crawford, T.L.; Bailey, L. et al. 1990. The world beef market: government intervention and multilateral policy reform. U.S. Dept. of Agriculture, Economic Research Service, Staff Report No. AGES9051, Rockville, MD.

Hall, S.M. 1985. BPA-CDSC surveillance of hemolytic uraemic syndrome 1983-84. Commun Dis Rep Wkly Edition 85:3-6.

Hancock, D.D. 1993. Myths and speculations about *E. coli* 0157:H7. Pop Med News 6:1-4.

Hancock, D.D.; Besser, T.E. and Rice, D.H. 1993a. Macro- and micro-epidemiology of *E. coli* 0157:H7: prospects for pre-harvest control. Submitted to Proceedings of U.S. Animal Health Assoc.

Hancock, D.D.; Wells, S.J.; Thomas, L.A. et al. 1993b. National prevalence study for *E. coli* 0157:H7 in United States dairy calves (unpublished).

Hancock, D.D.; Besser, T.E.; Kinsel, M. L. et al. 1994. The prevalence of *Escherichia coli* 0157:H7 in dairy and beef cattle in Washington state. Epidemiol Infect (in press).

Harris, A.A. 1990. Hemorrhagic colitis and *Escherichia coli* 0157:H7-identifying a messenger while pursuing the message. Mayo Clin Proc 65:884-888.

Herriott, D. 1993. Raw milk-associated *E. coli* 0157:H7 outbreaks. September 28, 1993 (unpublished).

Heyworth, B. and Brown, J. 1975. Jejunal microflora in malnourished Gambian children. Arch Dis Child 50:27-33.

Higginbotham, G.E. and Bath, D.L. 1993. Evaluation of *Lactobacillus* fermentation cultures in calf feeding systems. J Dairy Sci 76:615-620.

Hinton, A.; Spates, G.E.; Corrier, D.E. et al. 1991. In vitro inhibition of the growth of *Salmonella typhimurium* and *Escherichia coli* 0157:H7 by bacteria isolated form the cecal contents of adult chickens. J Food Protect 54:496-501.

Hoard's Dairyman. 1982. Continuing market study. Research department, Hoard's Dairyman, Fort Atkinson, WI.

Hoard's Dairyman. 1991. Continuing market study. Research department, Hoard's Dairyman, Fort Atkinson, WI.

Hoard's Dairyman. 1992. Continuing market study. Research department, Hoard's Dairyman, Fort Atkinson, WI.

Hoard's Dairyman. 1993. Continuing market study. Research department, Hoard's Dairyman, Fort Atkinson, WI.

Hockin, J. and Lior, H. 1987. Hemorrhagic colitis and hemolytic uremic syndrome caused by *E. coli* O157:H7 in Canada. Can Dis Wkly Rep 13-45:203-204.

Hockin, J.; Lior, H.; Mueller, L. et al. 1987. An outbreak of *E. coli* O157:H7 diarrhea in a nursing home - Alberta. Can Dis Wkly Rep 13-45:206.

Hockin, J.; Lior, H.; Stratton, F. et al. 1988. Incidence of hemorrhagic colitis due to *Escherichia coli* in Canada. Can Med Assoc J 139:1073-1074.

Hummel, R.; Tschape, H. and Witte, W. 1986. Spread of plasmid-mediated nourseothricin resistance due to antibiotic use in animal husbandry. J Basic Microbiol 26:461-466.

JAVMA. 1993. AVMA strengthens stand on milk pasteurization. J Am Vet Med Assoc 203:757.

Jay, J.M. 1986. Modern food microbiology, 3rd ed. Van Nostrand Reinhold Company, New York, NY.

Johnson, L.P.; Williams, S.E.; Neel, S.W. et al. 1994. Foodservice industry market profile study. Nutritional profile of foodservice ground beef. J Anim Sci (in press).

Jones, J.M. 1992. Food irradiation. In: Food Safety. Eagan Press, St. Paul, MN. pg. 301-330.

Kain, K.C.; Barteluk, R.L.; Kelly, M.T. et al. 1991. Etiology of childhood diarrhea in Beijing, China. J Clin Microbiol 29:90-95.

Khakhria, R.; Duck D. and Lior H. 1990. Extended phage-typing scheme for *Escherichia coli* 0157:H7. Epidemiol Infect 105:511-520.

Kim, K.-H.; Suh, I.-S.; Kim, J.M. et al. 1989. Etiology of childhood diarrhea in Korea. J Clin Microbiol 27:1192-1196.

Kim, M.S. and Doyle, M.P. 1992. Dipstick immunoassay to detect enterohemorrhagic *Escherichia coli* 0257:H7 in retail ground beef. Appl Environ Microbiol 58:1764-1767.

Kobayashi, K. and Harada, K. 1986. Enterohemorrhagic colitis associated with *Escherichia coli* 0157:H7 in Osako prefecture. In: Saito, M.; Nakaya, R. and Matsubara, Y. (eds.). Infectious enteritis in Japan. Saikon Shuppan. Showa, Tokyo, Japan, pg. 349-357.

Krause, K.R. 1992. The beef cow-calf industry, 1964-1987: location and size. U.S. Dept. of Agriculture, Economic Research Service, Agricultural Economic Report No. 659.

Lee, A. and Gemmell, E. 1972. Changes in the mouse intestinal microflora during weaning: role of volatile fatty acids. Infect Immun 5:1-7.

Lerman, Y.; Cohen, D.; Gluck, A. et al. 1992. A cluster of cases of *Escherichia coli* 0157 infection in a day-care center in a communal settlement (Kibbutz) in Israel. J Clin Microbiol 30:520-521.

Levy, S.B. and Marshall, B.M. 1988. Genetic transfer in the natural environment. In: Sussman, M.; Collins, C.H.; Skinner, S.K. et al. (eds.). The release of genetically engineered microorganisms. Academic Press, London, England, pg.61-67.

Line, J.E.; Fain, A.R. Jr.; Moran, A.B. 1991. Lethality of heat to *Escherichia coli* O157:H7: D-value and Z-value determinations in ground beef. J Food Protect 54:762-766.

Lopez, E.L.; Diaz, M.; Grinstein, S. et al. 1989. Hemolytic uremic syndrome and diarrhea in Argentine children: the role of Shiga-like toxins. J Infect Dis 160:469-475.

Lyons, R.W.; Samples, C.L.; DeSilva, H.N. et al. 1980. An epidemic of resistant *Salmonella* in a nursery: animal-to-human spread. J Am Med Assoc 243:546-547.

MacDonald, K.L.; O'Leary, M.J. and Cohen, M.L. 1988. *Escherichia coli* 0157:H7, an emerging gastrointestinal pathogen - results of a one-year, prospective, population-based study. J Am Med Assoc 259:3567-3570.

March, S.B. and Ratnam, S. 1986. Sorbitol-MacConkey medium for detection of *Escherichia coli* O157:H7 associated with hemorrhagic colitis. J Clin Microbiol 23:869-872.

Marshall, W.F.; McLimans, C.A.; Yu, P.K. et al. 1990. Results of a 6-month survey of stool cultures for *Escherichia coli* O157:H7. Mayo Clin Proc 65:787-792.

Martin, D.L.; MacDonald, K.L.; White, K.E. et al. 1990. The epidemiology and clinical aspects of the hemolytic uremic syndrome in Minnesota. N Engl J Med 323:1161-1167.

Martin, M.L.; Shipman, L.D.; Wells, J.G. et al. 1986. Isolation of *Escherichia coli* 0157:H7 from dairy cattle associated with two cases of haemolytic uraemic syndrome. Lancet 2:1043.

Mata, L.J.; Jimenez, F.; Cordon, M. et al. 1972. Gastrointestinal flora of children with protein-calorie malnutrition. Am J Clin Nutr 25:1118-1126.

McCoy, J.H. and Sarhan, M.E. 1988. Livestock and meat marketing. Van Nostrand Reinhold Company, Inc., New York, NY.

McGowan, K.L.; Wickersham, E. and Strockbine, N.A. 1989. *Escherichia coli* 0157:H7 from water. Lancet 29:967-968.

Meyers, J.A.; Sanchez, D.; Elwell, L.P. et al. 1976. Simple agarose gel electrophoretic method for the identification and characterization of plasmid deoxyribonucleic acid. J Bacteriol 27:1529-1537.

Mohammad, A.; Peiris, J.S.M. and Wijewanta, E.A. 1986. Serotypes of verocytotoxigenic *Escherichia coli* isolated from cattle and buffalo calf diarrhoea. FEMS Microbiol Lett 35:261-265.

Mohammad, A.; Peiris, J.S.M.; Wijewanta, E.A. et al. 1985. Role of verocytotoxigenic *Escherichia coli* in cattle and buffalo calf diarrhoea. FEMS Microbiol Lett 26:281-283.

Montenegro, M.A.; Bulte, M.; Trumpf, T. et al. 1990. Detection and characterization of fecal verotoxin-producing *Escherichia coli* from healthy cattle. J Clin Microbiol 28:1417-1421.

Montes, A.J. and Pugh, D.G. 1993. The use of probiotics in food animal practice. Vet Med 88:282-288.

Morishita, Y. and Miyaki, K. 1979. Effects of age and starvation on the gastrointestinal microflora and the heat resistance of faecal bacteria in rats. Microbiol Immunol 23:455-470.

Neel, S.W.; Williams, S.E.; Johnson, L.P. et al. 1994. Foodservice industry market profile study. Beef marketing overview and product distribution survey. J Foodservices (in press).

Neill, M.A. 1989. *E. coli* 0157:H7 - current concepts and future prospects. J Food Safety 10:99-106.

Neill. M.A.; Tarr, P.I.; Clausen, C.R. et al. 1987. *Escherichia coli* O157:H7 as the predominant pathogen associated with the hemolytic uremic syndrome: a prospective study in the Pacific Northwest. Pediatrics 80:37-40.

Nottingham, P.M. and Urselmann, A.J. 1961. *Salmonella* infection in calves and other animals. NZ J Ag Res 4:449-460.

O'Brien, A.D. and Holmes, R.K. 1987. Shiga and Shiga-like toxins. Micro Rev 51:206-220.

O'Brien, A.D.; LaVeck, G.D.; Thompson, M.R. et al. 1982. Production of *Shigella dysenteriae* type 1-like cytotoxin by *Escherichia coli*. J Infect Dis 1446:763-769.

O'Brien, A.D.; Chen, M.E.; Holmes, R.K. et al. 1984. Environmental and human isolates of *Vibrio cholerae* and *Vibrio parahaemolyticus* produce a *Shigella dysenteriae* 1 (Shiga)-like cytotoxin. Lancet 1:77-78.

Okrend, A.J.G.; Rose, B.E. and Matner, R. 1990. An improved screening method for the detection and isolation of *Escherichia coli* 0157:H7 from meat, incorporating the 3M Petrifilm Test Kit - HEC - for hemorrhagic *Escherichia coli* 0157:H7. J Food Protect 53:936-940.

Orskov, F.; Orskov, I. and Villar, J.A. 1987. Cattle as a reservoir of verotoxin-producing *Escherichia coli* O157:H7. Lancet 2:276.

Ostroff, S.M.; Kobayashi, J.M. and Lewis, J.H. 1989. Infections with *Escherichia coli* 0157:H7 in Washington state, the first year of statewide disease surveillance. J Am Med Assoc 262:355-359.

Ostroff, S.M.; Griffin, P.M.; Tauxe, R.V. et al. 1990. A statewide outbreak of *Escherichia coli* 0157:H7 infections in Washington state. Am J Epidemiol 132:239-247.

Padhye, N.V. and Doyle, M.P. 1992. *Escherichia coli* 0157:H7: epidemiology, pathogenesis, and methods for detection in food. J Food Protect 55:555-565.

Pai, C.H.; Kelly, J.K. and Meyers, G.L. 1986. Experimental infection of infant rabbits with verotoxin-producing *Escherichia coli*. Infect Immun 51:16-23.

Pai, C.H.; Ahmed, N.; Lior, H. et al. 1988. Epidemiology of sporadic diarrhea due to verocytotoxin-producing *Escherichia coli*: a two-year prospective study. J Infect Dis 157:1054-1057.

Pao, E.M.: Fleming, K.H.; Guenther, P.M. et al. 1982. Foods commonly eaten by individuals: amount per day and per eating occasion. U.S. Dept. of Agriculture, Human Nutrition Information Service, Home Economics Report Number 44, Washington, DC.

Parsonnet, K.C. and Kass, E.H. 1987. Does prolonged exposure to antibiotic-resistant bacteria increase the rate of antibiotic-resistant infection? Antimicrob Agents Chemother 31:911-914.

Pavia, A.T.; Nichols, C.R.; Green, D.P. et al. 1990. Hemolytic-uremic syndrome during an outbreak of *Escherichia coli* 0157:H7 infections in institutions for mentally retarded persons: clinical and epidemiologic observations. J Pediatrics 116:544-550.

Pawelzik, M. 1991. Pathogenic *Escherichia coli* O157:H7 and their detection. Acta Microbiologica Hungarica 38:315-320.

Perry, M.B.; Bundle, D.R.; Gidney, M.A. et al. 1988. Identification of *Escherichia coli* serotype 0157 strains by using a monoclonal antibody. J Clin Microbiol 26:2391-2394.

PHLS Communicable Disease Surveillance Center. 1990. Haemolytic uraemic syndrome surveillance: British paediatric surveillance unit/CDSC surveillance scheme. Commun Dis Rep Wkly Edition 90:1.

Pierard, D.; Van Etterijck, R.; Breynaert, J. et al. 1990. Results of screening for verocytotoxin-producing *Escherichia coli* in faeces in Belgium. Eur J Clin Microbiol Infect Dis 9:198-201.

Potter, M.E.; Blaser, M.J.; Sikes, R.K. et al. 1983. Human *Campylobacter* infection associated with certified raw milk. Am J Epidemiol 117:475-483.

Pryor, W.; Hodson, E.; McIntyre, P. et al. 1990. Toxigenic *Escherichia coli* in haemolytic-uraemic syndrome. Med J Aust 152:221-222.

Rasmussen, M.A.; Cray, W.C.; Casey, T.A. et al. 1993. Rumen contents as a reservoir of enterohemorrhagic *Escherichia coli*. FEMS Microbiol Lett 114:79-84.

Ratnam, S.; March, S.B. and Sprague, W.D. 1986. Are humans a source of *Escherichia coli* 0157:H7, the agent of hemorrhagic colitis? N Engl J Med 315:1612-1613.

Remis, R.S.; MacDonald, K.L.; Riley, L.W. et al. 1984. Sporadic cases of hemorrhagic colitis associated with *Escherichia coli* 0157:H7. Ann Intern Med 101:624-626.

Renwick, S.A.; Wilson, J.B.; Clarke, R.C. et al. 1993. Evidence of direct transmission of *Escherichia coli* O157:H7 infection between calves and a human. J Infect Dis 168:792-793.

Rice, E.W.; Johnson, C.H.; Wild, D.K. et al. 1992. Survival of *Escherichia coli* O157:H7 in drinking water associated with a waterborne disease outbreak of hemorrhagic colitis. Letters in Appl Microbiol 15:38-40.

Riley, L.W. 1987. The epidemiologic, clinical, and microbiologic features of hemorrhagic colitis. Ann Rev Microbiol 41:383-407.

Riley, L.W.; Remis, R.S.; Helgerson, S.D. et al. 1983. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. N Engl J Med 308:681-685.

Robinson, D.A. and Jones, D.M. 1981. Milk-borne *Campylobacter* infection. Brit Med J 282:1374-1376.

Robinson, R.A. 1967. *Salmonella* excretion by sheep following yarding. NZ Vet J 15:24-25.

Robinson, R.A. and Royal, W.A. 1971. Field epizootiology of *Salmonella* infection in sheep. NZ J Ag Res 14:442-456.

Rogers, J.A. and Davis, C.L. 1982. Rumen volatile fatty acid production and nutrient utilization in steers fed a diet supplemented with sodium bicarbonate and monensin. J Dairy Sci 65:944-952.

Rolfe, R.D. 1991. Population dynamics of the intestinal tract. In: Blankenshipp, J.S.; Cox, N.A.; Craven, S.E. et al. (eds.). Colonization control of human bacterial enteropathogens in poultry. Academic Press, Inc., New York, NY, pg. 59-76.

Ronner, A.B. and Cliver, D.O. 1990. Isolation and characterization of a coliphage specific for *Escherichia coli* 0157:H7. J Food Protect 53:944-947.

Rowe, B.; Threlfall, E.J.; Ward, L.R. et al. 1979. International spread of multiresistant strains of *Salmonella typhimurium* phage types 204 and 193 from Britain to Europe. Vet Rec 105:468-469.

Rowe, P.C.; Orrbine, E.; Wells, G.A. et al. 1991. Epidemiology of hemolytic-uremic syndrome in Canadian children from 1986 to 1988. J Pediatr 119:218-224.

Ryan, C.A.; Tauxe, R.V.; Hosek, G.W. et al. 1986. *Escherichia coli* 0157:H7 diarrhea in a nursing home: clinical, epidemiological, and pathological findings. J Infect Dis 154:631-638.

Ryan, C.A.; Nickels, M.C.; Hargett-Bean, N.T. et al. 1987. Massive outbreak of antimicrobial-resistant salmonellosis traced to pasteurized milk. J Am Med Assoc 258:3269-3274.

Salisbury, R.M. 1958. *Salmonella* infections in animals and birds in New Zealand. NZ Vet J 6:76-86.

Savage, D.C. 1982. The effect of stress, diet and environment on the stability of the gastrointestinal microflora. In: White, E.G. (ed.). Normal and induced changes in the gastrointestinal microflora in man and animals with special regard to animal performance. Proc Internat Symposium, Oslo, Norway, September, 1980. Verlag Paul Parey, Berlin, Germany, pg. 23-31.

Savell, J.W.; Harris, J.J.; Cross, H.R. et al. 1991. National beef market basket survey. J Anim Sci 9:2883-2893.

Schaedler, R.W.; Dubos, R. and Costello, R. 1965. The development of the bacterial flora in the gastrointestinal tract of mice. J Exp Med 122:59-66.

Scheuermann, S.E. 1993. Effect of the probiotic Paciflor (CIP5832) on energy and protein metabolism in growing pigs. Anim Feed Sci & Tech 41:181-189.

Sekla, L.; Milley, D.; Stackiw, W. et al. 1990. Verotoxin-producing *Escherichia coli* in ground beef in Manitoba. Can Med Assoc J 143:519-521.

Sharpe, J.C.M. 1989. Milk-borne infection. J Med Microbiol 29:239-242.

Shimada, T.; Kosako, Y.; Isshiki, Y. et al. 1992. Enterohemorrhagic *Escherichia coli* O157:H7 possesses somatic (O) antigen identical with that of *Salmonella* $O30_1$. Current Micro 25:215-217.

Siragusa, G.R. and Nettles, C.G. 1993. Efficacy of organic acid decontamination of *Escherichia coli* 0157:H7 on beef carcass tissue in a pilot scale model carcass washing system. Summary report prepared by U.S. Dept. of Agriculture, Agricultural Research Service, U.S. Meat Animal Research Center, Clay Center, NE.

Smith, H.W. 1965. Observations on the flora of the alimentary tract of animals and factors affecting its composition. J Pathol Bacteriol 89:95-122.

Smith, H.W. and Jones, J.E.T. 1963. Observations on the alimentary tract and its bacterial flora in healthy and diseased pigs. J Pathol Bacteriol 86:387-412.

Smith, H.R.; Rowe, B.; Gross, R.J. et al. 1987. Haemorrhagic colitis and verocytotoxin-producing *Escherichia coli* in England and Wales. Lancet 1:1062-1064.

Speirs, J.I. and Stavric, S. 1989. *Escherichia coli* associated with hemorrhagic colitis. Can Inst Food Sci Technol J 22:205-208.

Spika, J.S.; Parsons, J.E.; Nordenberg, D. et al. 1986. Hemolytic uremic syndrome and diarrhea associated with *Escherichia coli* 0157:H7 in a day care center. J Pediatrics 109:287-291.

Spika, J.S.; Waterman, S.H.; Soo Hoo, G.W. et al. 1987. Chloramphenicol-resistant *Salmonella newport* traced through hamburger to dairy farms. N Engl J Med 316:565-570.

Sramkova, L.; Bielaszewska, M.; Janda, J. et al. 1990. Vero cytotoxin-producing strains of *Escherichia coli* in children with haemolytic uraemic syndrome and diarrhoea in Czechoslovakia. Infection 18:204-209.

Stavric, S. 1992. Defined cultures and prospects. Intl J Food Microbiol 15:245-263.

Stavric, S.; Buchanan, B. and Gleeson, T.M. 1992. Competitive exclusion of *Escherichia coli* 0157:H7 from chicks with anaerobic cultures of faecal microflora. Letters in Appl Microbiol 14:191-193.

Strockbine, N.A.; Marques, L.R.M.; Holmes, R.K. et al. 1985. Characterization of monoclonal antibodies against Shiga-like toxin from *Escherichia coli*. Infect Immun 50:695-700.

Suthienkul, O.; Brown, J.E.; Seriwatana, J. et al. 1990. Shiga-like toxin-producing *Escherichia coli* in retail meats and cattle in Thailand. Appl Environ Microbiol 56:1135-1139.

Swerdlow, D.L.; Woodruff, B.A.; Brady, R.C. et al. 1992. A waterborne outbreak in Missouri of *Escherichia coli* 0157:H7 associated with bloody diarrhea and death. Ann Intern Med 117:812-819.

Szabo, R.; Todd, E.; MacKenzie, J. et al. 1990. Increased sensitivity of the rapid hydrophobic grid membrane filter enzyme-labeled antibody procedure for *Escherichia coli* 0157 detection in foods and bovine feces. Appl Environ Microbiol 56:3546-3549.

Tannock, G.W. 1983. Effect of dietary and environmental stress on the gastrointestinal microbiota. In: Hentges, D.J. (ed.). Human intestinal microflora in health and disease. Academic Press, Inc., New York, NY, pg. 517-539.

Tannock, G.W.; McInnes, H.; Shirley, G. et al. 1971. Salmonellosis in sheep wintered outdoors on straw and sawdust pads. NZ Vet J 19:29-32.

Tarr, P.I.; Neill, M.A.; Allen, J. et al. 1989. The increasing incidence of the hemolytic-uremic syndrome in King County, Washington: lack of evidence for ascertainment bias. Am J Epidemiol 129:582-586.

Timoney, J.F.; Gillespie, J.H.; Scott, F.W. et al. (eds.). 1988. Hagan and Bruner's Microbiology and Infectious Diseases of Domestic Animals. Comstock Publishing Associates, Cornell University Press, Ithaca, NY. pg. 61-73.

Todd, E.C.D.; Szabo, R.A.; Peterkin, P. et al. 1988. Rapid hydrophobic grid membrane filter-enzyme labeled antibody procedure for identification and enumeration of *Escherichia coli* O157 in foods. Appl Environ Microbiol 54:2536-2540.

Tyler, J.W.; Wolfe, D.F. and Maddox, R. 1992. Clinical indications for dietary ionophores in ruminants. The Compendium - Food Anim 14:989-993.

Tzipori, S.; Wachsmuth, I.K.; Chapman, C. et al. 1986. The pathogenesis of hemorrhagic colitis caused by *Escherichia coli* O157:H7 in gnotobiotic piglets. J Infect Dis 154:712-716.

USDA:APHIS. 1993. Bovine spongiform encephalopathy: implications for the United States. U.S. Dept. of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, Centers for Epidemiology and Animal Health, Fort Collins, CO.

USDA:APHIS. 1994. A survey of cattle packers with application to the production of ground beef. U.S. Dept. of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, Centers for Epidemiology and Animal Health, Fort Collins, CO.

USDA:ERS. (a). Livestock and poultry situation and outlook. U.S. Dept. of Agriculture, Economic Research Service, Commodity Economics Division, Washington DC, various issues.

USDA:ERS. (b). Foreign agricultural trade of the United States. U.S. Dept. of Agriculture, Economic Research Service, Commodity Economics Division, Washington, DC, various issues.

USDA:ERS. 1993. Food safety issues: modernizing meat inspection. U.S. Dept. of Agriculture, Economic Research Service, Ag Outlook, Washington, DC.

USDA:FAS. 1992. Dairy, livestock, and poultry: world livestock situation. U.S. Dept. of Agriculture, Foreign Agricultural Service, FAS Circular Series: FL&P 4-92, Washington, DC.

USDA:FSIS. Animal disposition reporting system. U.S. Dept. of Agriculture, Food Safety and Inspection Service, Washington, DC.

USDA:FSIS. 1994. Nationwide beef microbiological baseline data collection program: steers and heifers October 1992 - September 1993. U.S. Dept. of Agriculture, Food Safety and inspection Service, Science and Technology, Microbiology Division, Washington, DC.

USDA:HNIS. 1993. Food and nutrient intakes by individuals in the United States, 1 day, 1987-88. Nationwide food consumption survey 1987-88, NFCS Rep No. 87-I-1, U.S. Dept. of Agriculture, Human Nutrition Information Service, Hyattsville, MD.

USDA:NASS. (a). Agricultural statistics. U.S. Dept. of Agriculture, National Agricultural Statistics Service, Washington, DC, various issues.

USDA:NASS. (b). Livestock slaughter. U.S. Dept. of Agriculture, National Agricultural Statistics Service, Washington, DC, various issues.

USDA:NASS. (c). Cattle on feed. U.S. Dept. of Agriculture, National Agricultural Statistics Service, Washington, DC, various issues.

USDA:NASS. 1990. Milk: final estimates 1983-87. U.S. Dept. of Agriculture, National Agricultural Statistics Service, Agricultural Statistics Board, Washington, DC.

USDA:NASS. 1993a. Cattle. U.S. Dept. of Agriculture, National Agricultural Statistics Service, Agricultural Statistics Board, Washington, DC.

USDA:NASS. 1993b. Milk production, disposition, and income: 1992 summary. U.S. Dept. of Agriculture, National Agricultural Statistics Service, Agricultural Statistics Board, Washington, DC.

USDA:OE. 1992. Possible effects of NAFTA on the U.S. beef and pork industries. U.S. Dept. of Agriculture, Office of Economics, Washington, DC.

USDA:P&SA. Packers and stockyards statistical report. U.S. Dept. of Agriculture, Packers and Stockyards Administration, Washington, DC, various issues.

USDC:BC. 1989. 1987 Census of agriculture: geographic area series, Vol. 1, Part 51, U.S. Dept. of Commerce, Bureau of the Census. Washington, DC.

Walker, C.W.; Upson, R. and Warren, R.E. 1988. Haemorrhagic colitis: detection of verotoxin producing *Escherichia coli* 0157 in a clinical microbiology laboratory. J Clin Pathol 41:80-84.

Wallace, J.M. 1980. Milk-associated *Campylobacter* infection. Hlth Bull 38:57-61.

Wells, J.G.; Shipman, L.D.; Greene, K.D. et al. 1991. Isolation of *Escherichia coli* serotype O157:H7 and other Shiga-like-toxin-producing *E. coli* from dairy cattle. J Clin Microbiol 29:985-989.

Whittam, T.S.; Wolfe, M.L.; Wachsmuth, I.K. et al. 1993. Clonal relationships among *Escherichia coli* strains that cause hemorrhagic colitis and infantile diarrhea. Infect Immun 61:1619-1629.

Wierup, M. 1993. Information paper: control of *Salmonella* in food producing animals. Presented at the NVI/WHO International Course on *Salmonella* Control in Animal Production and Products, Malmo, Sweden.

Williams, L.P. and Newell, K.W. 1968. Sources of salmonellas in market swine. J Hyg 66:281-293.

Wuthe, H.H. 1987. *Escherichia coli* 0157:H7 bei hemoraghagischer colitis (letter). Disch Med Woehenschr 112:1761-1762.